Friedberger & Fröhner's Veterinary Pathology

[Authorised Translation]

TRANSLATED BY

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WITH NOTES ON BACTERIOLOGY

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CHAPTER I.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NASAL CAVITY.

ACUTE NASAL CATARRH OF HORSES. CORYZA.

Etiology.—The causes of acute nasal catarrh, or superficial inflammation of the mucous membranes of the nasal cavity are various.

1. *Primary*, independent, acute nasal catarrh is most frequently produced by cold, especially in spring and autumn. Limp, pampered and youthful animals are particularly predisposed to it. The much greater frequency of acute nasal catarrh in horses than in other animals, such as cattle, may be explained partly by their greater exposure to cold, and partly by the extensive width of their nostrils, whereby the mucous membranes are more exposed to external irritants. Among the latter we may mention, the inhalation of dust from the streets or from fodder, of smoke, fungi, spores, etc., which may be floating in the air after distribution of food, also from the ammoniacal air of close and crowded stables. The possibility of infectious influences in the production of coryza cannot be denied, as its occasional epizoötic appearance points clearly to some such cause. But nothing positive is as yet known on this head.

2. Acute nasal catarrh occurs *secondarily* with very great frequency as an accompaniment of other diseases, especially of infectious diseases and diseases of the breathing organs. Sometimes the catarrhal process extends from a primarily affected mucous membrane, as, *e.g.*, from the pharynx to the nasal cavity.

Symptoms.—The first signs of acute nasal catarrh consist in a diffuse hyperæmia (less frequently petechial), as well as in dryness of the nasal mucous membranes. The expired air is somewhat hotter, owing to the membrane being more fully

charged with blood (stage of inflammatory irritation). A few days later the chief symptom of the disease appears, viz., discharge from the nose; the mucous membrane is now shiny, swollen and often quite œdematously saturated (stage of secretion). The nasal discharge, which is usually bilateral, is at first serous, as clear as water, and of very thin, watery consistence; and in slighter cases this appearance does not change. But if the catarrhal process goes forward, the nasal outflow, owing to increased mucous secretion, becomes glairy, slimy, and also opaque and turbid, this arising from simultaneous active desquamation of epithelium. Lastly, the secretion becomes pus-like (serous, mucous, purulent catarrh) owing to the admixture of numerous white blood-corpuscles. Horses often snort, and the vicinity of the nostrils is usually found encrusted with dry discharges of mucous and pus-like character. Occasionally the lymphatic-glands of the throat are found slightly swollen.

The *duration* of acute nasal catarrh averages from eight to fourteen days. Recovery usually follows without any assistance, the discharge gradually abating. The disease may, however, from the outset be accompanied by severer signs, such as shivering fits, loss of appetite, slight rise of temperature and of pulsation, and may spread to neighbouring mucous membranes, such as the frontal sinus, or, by way of the lachrymal ducts, to the eyes. Then great heaviness is observed in the head, catarrhal conjunctivitis, watering of the eyes and avoidance of the light; very often this symptom is accompanied by laryngitis, stomatitis, catarrh of the superior maxillary sinus, etc. Finally, the condition may become chronic.

Therapeutics.—Treatment is necessary only in more pronounced cases; lighter ones recover just as quickly of themselves. The most effectual means is the local treatment of the nasal mucous membranes by inhalation of steam and hot vapour, to which we may add a little creolin or carbolic acid. It is hardly necessary in acute catarrh to rinse the nasal cavities with astringent fluids. Internally anti-catarrhal salts (neutral salts, common salt) may be administered in combination with aromatic vegetable powders. More important is good ventilation of the stable and bringing the animal out of doors, if the weather be favourable; moreover, a supply of green fodder, turnips, etc., or free grazing in the open, are all commended.

CHRONIC NASAL CATARRH OF HORSES.

Etiology.—In contrast to the acute form just described, chronic, primary nasal catarrh is much rarer among horses. It is developed from the acute by greater intensity and diffusion of the latter, and by longer and repeated operation of the causes. More common and much more important clinically, is the secondary nasal catarrh caused by other morbid processes. Of such we must especially mention: 1. Glanders, which often exhibits merely a chronic nasal catarrh. For this reason chronic nasal catarrh is always a very suspicious sign in horses, and requires watching with great care. 2. Catarrh of the nasal accessory cavities (superior maxillary sinus, frontal sinus) often reveals itself only by a chronic nasal discharge. 3. Tumours, parasites, and foreign bodies in the nasal cavities, necrosis of the turbinated bones. 4. Diseases of the teeth, especially suppurative periostitis of the alveoli of the teeth, with their complications. 5. Chronic affections of the breathing apparatus in general, as well as chronic constitutional diseases, such as leucæmia and anæmia.

Symptoms.—During chronic nasal catarrh the mucous membranes of the nose are pale or cyanotic and, if prolonged, they become thickened and are traversed by dilated veins. The nasal discharge is sometimes glairy, slimy and like the spawn of frogs, or it is putrescent, yellowish or dirty-grey in colour, smearing the nostrils, and occasionally smelling badly. Frequently one may perceive a streak upon the skin beneath the nasal opening and corresponding to the point at which the catarrhal secretion is discharged, upon which the skin-pigment is lacking. The quantity of the discharge varies much, it may grow to a veritable stream of mucus (blennorrhœa). Sometimes it ceases entirely for a while; and again a very great quantity may be discharged suddenly, as after movement, or on lowering the head. When the flow is from both nostrils, it generally indicates a primary catarrh; when from one only, a secondary (glanders, new growths, foreign bodies, necrosis of the turbinated bones in the nasal cavity, dental trouble, etc.); with the latter we often find also swelling and induration of the sub-maxillary glands.

As a peculiar complication of chronic nasal catarrh, when much prolonged, we sometimes find on the nasal septum,

so-called erosion-ulcers which are flat, superficial losses of substance, from a pin-head to a lentil-seed in size and with sharp, unthickened edges. Similar lesions are sometimes found after prolonged douching of the nasal fossæ and adjacent cavities with solutions of carbolic acid, creolin, lysol, and bazillol. These are sufficiently distinguishable from the ulcers of glanders by this description. Nevertheless, we must state that such erosion-ulcers occur also in glanders, as we have had occasion several times to observe, because the latter gives rise secondarily to chronic nasal catarrh; their presence therefore affords no presumption against the existence of glanders. The erosion-ulcers (formerly known as catarrhal ulcers) heal without leaving any cicatrix, by which also they may be known from true glanderous ulcers.

The *duration* of chronic nasal catarrh may extend to weeks, months, and even years, and the malady is in general difficult to cure. When much prolonged, hyperplastic processes may develop upon the nasal mucous membrane.

Diagnosis.—It is always very difficult, and sometimes impossible, to decide the question, so important for prognosis, whether we are dealing with a primary or a secondary chronic nasal catarrh. The best guide is often afforded by the course taken by the disease, which in a primary catarrh is more favourable than in a secondary. The fact also that the discharge is from both sides, may help to decide in favour of the primary form. But in any case, careful and frequent examination of the nasal cavity with a rhinoscope or a Pristley lamp is indispensable. For a veterinary hospital we recommend the special rhinoscope of Polansky and Schindelka, which renders it possible to examine fully the entire nasal mucous membrane of a horse. In many cases it is necessary to trephine the cavities of the nose, the frontal or superior maxillary sinuses, or experimentally to inoculate other animals with the catarrhal secretion, and this particularly if there be any suspicion of glanders. Compare on this point the paragraph on the differential diagnosis of glanders.

Therapeutics.—In this case also the treatment is essentially local, and consists either in the application of vapours of water, creolin, lysol, carbol, creosote, tar and oil of turpentine, which may be used one after the other in the order given, applying for a time steam only, and then the stronger vapour of carbol, etc.; or else in washing the nasal mucous membrane

with astringent liquids (1 to 2 per cent. solution of sulphate of zinc or creolin, or a ½ per cent. solution of nitrate of silver, a one-per-thousand solution of sublimate, etc.). These are either sprayed in through the nostrils, or introduced into the nasal cavity from above after trephining (Dieckerhoff). The adoption of trephining should not be postponed, especially in cases of unilateral chronic rhinitis, accompanied by scaly necrosis or the presence of foreign bodies. It is also of great service to rinse the nasal mucous membrane by means of a stout india-rubber tube or elastic stomach sound introduced through one of the posterior nasal openings. The value of internal remedies (ammonia, spirits of turpentine) is here very doubtful. But from a hygienic standpoint it is well to provide as fresh an atmosphere as possible, frequent sojourn in the open, cleanliness in the nostrils, manger, etc., with good fodder and attention to the skin. Finally, it is urgently advised to separate all horses suffering from chronic nasal catarrh as a precaution. Any contingent treatment of the erosion-ulcers with drugs is to be avoided, as they are thereby hindered in their development and considerably altered, so that the most important guide in diagnosis of glanders is removed.

Necrosis of the turbinated bones.—After chronic suppurative rhinitis (chronic nasal catarrh, strangles, petechial fever), also in connection with fracture of the nasal bones and diseases of the teeth, a suppurative, necrotic inflammation of the turbinated bones, especially of the anterior nasal turbinal often develops in a horse. This is characterised by a chronic, unilateral, purulent or ichorous, fœtid, copious nasal discharge; unilateral enlargement of the cervical lymphatic glands, and sometimes by unilateral nasal dyspnœa. Similar processes also frequently occur in the ethmoid bone, and on the nasal septum. The treatment consists in removal of the necrotic pieces and the pent-up purulent matter, after one or several trephinings of the nasal fossæ, followed by antiseptic irrigation.

NASAL CATARRH IN SHEEP.

General Remarks.—The nasal catarrh of sheep has in so far an especial importance as that hitherto quite obviously different diseases have been included under the name of its synonyms, "sheep-glanders," and "benign" and "malignant catarrhal fever." The last of these should be regarded as an infectious disease, in which the affection of the nasal membranes represents only a symptom. We distinguish, on this account, the following varieties of nasal catarrh:

1. *Simple acute nasal catarrh* is in sheep of so slight a

covered and surrounded by a membrane-like deposit, which can be removed with difficulty only or not at all. When it falls off, little flat, shallow ulcers with very red edges, which lie close together in great numbers, are found in place of the nodules. These ulcers quickly heal without leaving any scar, their base being covered with epithelium growing from the edges. Nodules and ulcers of similar description appear also on the borders of the nose, around the nostrils, and also on the skin of the upper lip and lower edge of the messeter. From these ulcers inflammatory strands of the lymph-vessels often proceed to the much swollen lymph-glands of the throat, even to those of the neck and shoulder or anterior breast. We have observed swellings as thick as an arm, and corresponding to the course of the larger superficial lymph-vessels, along the mastoido-humeralis muscle to the entrance of the breast. These swellings of the large lymph-vessels of the neck are hard and tense, like those on the head, and sometimes form abscesses. We also noticed in many cases conjunctivitis and even decided blennorrhœa. The *course* of the malady is mostly benign, and complete recovery usually follows in from two to four weeks. Any confusion with glanders is scarcely possible with careful examination. The superficial and always numerous ulcers, which readily heal and leave no scar behind; the frequent spread of the same upon the outer skin; the presence of inflammation in the lymph-vessels; and the benign course of the disease, all assure diagnosis. An examination of the oral cavities will guard against the not infrequent confusion with stomatitis contagiosa pustulosa; these cavities are almost always attacked in the latter disease. *Treatment* is inhalation of vapour of water, creolin and carbol. It is well also to rub the swollen lymph glands and vessels with an ointment of iodoform, creolin, camphor, mercury or carbol. Direct treatment of the ulcers by touching them with nitrate of silver we consider unnecessary, and under some circumstances, as rendering a differentiation from glanders more difficult.

2. **Croupous Inflammation of the Nasal Mucous Membrane** arises first from violent irritation of the membrane, as for instance, from inhaling smoke and hot air on an outbreak of fire. Strangles also may be accompanied by croupy rhinitis, but this is rare. Further, nasal croup may occur among horses, especially cavalry remounts, and sometimes enzoötically, which gives much probability to the assumption of

an infective cause. This form of croupous rhinitis must therefore be regarded as an infectious disease. Röll and Buckmüller, who frequently observed the disease in Vienna, describe its symptoms as consisting in a deposit upon the nasal mucous membrane of grey or reddish yellow, skin-like, croupy membrane, which is sometimes of considerable extent, and by suppuration is gradually pushed off. The subjacent mucous membrane is extremely hyperæmic, and, after the croupy membrane has been cast off, appears excoriated and bleeds readily. At the same time there is a viscid, yellowish nasal discharge, and, in severer cases, difficulty of breathing, fever, sensitive swelling of the lymphatic vessels of the upper lip, cheeks and lower jaw, as well as of the glands in the cavity of the latter. *Prognosis* is usually favourable, and *treatment* the same as for simple nasal catarrh. Owing to its infectious character, sick animals should be isolated. A similar infectious nasal croup has been occasionally seen in oxen. In contrast to malignant catarrhal fever, it is very benign and never complicated with affection of the eyes. It affects several inmates of the same stable at one time with high fever, their breathing becoming very noisy, but in about eight days they have mostly recovered. Berndt saw this singular infective malady in 16 cows of one byre.

3. **Diphtheritic Inflammation of the Nasal Mucous Membrane** has nothing in common with human diphtheria. It is rather to be regarded as merely a necrotic inflammation of the mucous membrane, which is rare as a primary disease, and is noticed, for instance, after the action of certain irritants, mechanical, thermic, chemical and infectious (necrosis-bacillus). It is seen more frequently as a secondary accompaniment of several infectious diseases, as in acute glanders, the petechial fever of horses and malignant catarrhal fever of cattle, after consuming fodder infected with fungi. See the chapter on Diphtheria, and text book on Surgery, the chapter Necrosis.

4. **Phlyctænular Inflammation of the Nasal Mucous Membrane** occurs most frequently as a co-symptom of strangles (as so-called phylctænular strangles), also in very severe, simple acute nasal catarrh. It is nothing but a vesicular catarrh, in which small blisters rise upon the nasal membrane, at first clear like water, but their contents may later become purulent. By the bursting of these blisters and drying up of their contents,

thin scabs are formed, which fall off when the tissue is completely renewed.

NEW GROWTHS IN THE NASAL CAVITY.

1. **Fibromata** *or myxomata* (*myxo-fibromata*) occur either as localised connective-tissue tumours, or as so-called nasal polypi, either pedunculated or upon a broad base, or else they form flat, chronic proliferations of mucous membrane. Polypous fibromata, which are sometimes visible even from without, keep up a chronic nasal catarrh and cause a wheezing, snoring sound by narrowing the affected nasal passage. The breath can often pass through one nostril only, so that, on placing the hand in front of the diseased nostril, no air-current, or only a very faint one, can be perceived, and by closing the sound nostril dyspnœa is at once caused. Occasionally we may see superficial necrosis and an ichorous nasal discharge with swelling of the glands upon one side. As the polypi grow, they may proliferate into the cavities of the mouth and pharynx, or press the septum to the other side. The flat proliferations of mucous membrane produce a lumpy surface upon it and, like the polypus form, are followed by chronic catarrh, stenosis of the nasal passages, difficulty of breathing, and sometimes ulcerative decomposition with an unpleasant nasal discharge.

2. **Adeno-fibromata** (erroneously styled *Rhinosclerom*), are not uncommon in horses on both sides of the lower third of the nasal membrane (the epithelium of the cutis), forming multiple hard and raised yellow-red tumours in size from a lentil to a walnut, which are generally ulcerated and crusted with blood and frequently cause nasal dypsnœa and bleeding from the nose.

3. **Sarcomata** and **Carcinomata** are rare. They usually displace the neighbouring bones (of the nose, maxilla and palate), and may even up-lift the facial bones, forcing themselves into the oral and pharyngeal cavities, where they cause loosening of the back teeth, difficulty of chewing and swallowing. They also often become ichorous, so that a foul-smelling, discoloured discharge from one nostril is caused, with swelling of the submaxillary glands on that side. Carcinomata also produce bleeding at the nose (a source of confusion with glanders). The starting-point of sarcomata is usually the

periosteum of the adjoining bones (osteo-sarcoma). Metastatic formations in the sub-maxillary lymph-glands are in most cases observed along with carcinomata and sarcomata.

4. **Angiomata** of the nasal septum arise from dilatation and new growth of vessels, and in most cases assume a cavernous character (angioma cavernosum). They are never sharply delimited, but extended superficially, so that they sometimes attain the size of the palm of the hand, their colour being dark brown or bluish-red. After mechanical irritation or increased blood-pressure caused by violent exertion, they easily bleed, so that periodic bleeding at the nose results (in one case this has been observed in the horse during ten years); they show also a great tendency to ulceration. The ulcers are often covered with blood-coagula and have a reddish-brown base. When they heal, scars remain, as from the ulcers of glanders. Moreover, on ulceration of an angioma, a bloody or discoloured nasal discharge begins, with swelling in the submaxillary glands. As will be seen from this description, angiomata may be only too easily confused with glanders; and in many cases diagnosis is assured only by microscopical examination of the new growths.

5. As rarer forms of new growth we would mention: lipomata, bothryomycomata, enchondromata, odontomata, osteomata, adenomata, actinomycomata, tuberculous new growths, dermoid cysts, colloid cysts, tumours which contain strongylus armatus, and also, in dogs, the occurrence of hair upon the mucous membrane of the nose. Congenital hypertrophy of the nasal bones has been also observed.

BLEEDING FROM THE NOSE. EPISTAXIS.

Etiology.—Bleeding from the nose is only a symptom, and consequently just as little an independent disease as hæmaturia. It occurs in all animals, but is most common in the horse. Its causes may be of a greatly varied nature, and during life very often cannot with certainty be determined. The most important affections of the nasal mucous membranes are:

1. *Traumatic influences*, injuries, contusions, fractures of the bones.

2. *Hyperæmia* and inflammation of the nasal mucous membrane.

3. *New growths* (especially adeno-fibromata and angiomata), and ulcers (glanders).

4. *Abnormal blood-pressure* and especially the disturbances of the circulation following violent exertion (racing), also inflammation of the brain, heart failure, lung affections, etc.

5. *General diseases with hæmorrhagic diathesis* (strangles, hæmophilia, petechial fever, anthrax, leucæmia, etc.). In horses a periodical and usually unexpected bleeding at the nose is often a symptom of glanders. Such bleeding in horses is therefore always a serious symptom.

Respecting bleeding from the nose in dogs, compare the chapter on dochmiasis (Vol. I., p. 257).

Symptoms.—In epistaxis the discharge is usually from one nostril only, seldom from both; sometimes in drops, at others in a slender stream. Not unfrequently the usual nasal discharge is merely streaked with blood. The bleeding is often only temporary, or it may be repeated several times; but in other cases it continues till the animal bleeds to death. In contrast to hæmorrhage from the lungs, the blood is not frothy, and there is no cough.

Therapeutics.—When the bleeding is slight, no treatment is required. In severer cases we try first of all injections of styptic solutions into the nasal cavity, especially a 5 to 10 per cent. solution of perchloride of iron. If the bleeding be dangerous, the affected nostril must be plugged with cotton-wool soaked in creolin, carbol or sublimate, for which purpose, under some circumstances, the nasal cavity must be trephined. Styptic remedies may at the same time be internally administered: sugar of lead, tannin, sulphate of iron, ergot or hydrastis. The subcutaneous and intravenous injection of gelatine, recently recommended for arrest of hæmorrhage, should be discarded as too dangerous.

CHAPTER II.

DISEASES OF THE ACCESSORY CAVITIES OF THE NOSE.

Inflammation of the Maxillary and Frontal Sinuses of Horses.

Occurrence.—Among the accessory cavities of the nose the superior maxillary sinus is most frequently attacked by catarrh, particularly its upper and larger division, less so the lower part and the frontal sinus, and still more rarely the ethmoid cells and sphenoidal cavities. It is usually unilateral.

Etiology.—Catarrh of the sinuses, which usually runs a chronic course, may be first caused by traumatic influences (fracture or infraction of the bone), perhaps also by cold, but it more frequently arises from the spread of inflammation from the nasal cavity (particularly in glanders) or from the alveoli of the molar teeth (alveolar periostitis). But chronic catarrh of the mucous membrane concerned may be produced by a new growth, such as sarcoma or carcinoma, and less often myxoma, botryomycoma, also foreign bodies, such as pieces of hay or straw, fragments of bone, bits of gauze, tow, and wadding, entrance of mould-fungi or echinococci in the nasal cavity. Glanders also spreads sometimes to the mucous membrane of the superior maxillary sinus. Rickets may in rarer cases be the origin of the catarrh in foals.

Anatomical Conditions.—At the outbreak of the complaint the mucous membranes of these accessory cavities are swollen, of a bluish or dark red colour and very highly charged with blood. Between the membranes and the bone we find a yellowish gelatinous exudation; while both bones and external skin are over-filled with blood. If the process be prolonged, the mucous membrane thickens and gives off a fluid, at first serous and later mucous and purulent, which, owing to the

swelling up of the orifice of discharge in the middle meatus of the nose, accumulates, thickens and frequently, also, decomposes (retention-dropsy or empyema of the frontal and superior maxillary sinus). In exceptional cases concretions form. Matter also sometimes collects between the mucous membrane and the bone, as a result of which the bone is wasted and thinned. In this way the external bony cover is bulged outwards and the cavity itself enlarged; the septum between the two divisions of the maxillary sinus may also become attenuated and even disappear entirely. In exceptional cases the catarrhal products of inflammation may even break through and form a fistula, or the pus may make itself a way to the dental alveoli and produce caries of the teeth. On the contrary a communication may be established between the spaces left by defective teeth and the sinus; when we sometimes find the sinus stuffed with fodder. The enlargement of the frontal sinus may, moreover, lead to constriction of the brain-cavity. In a similar manner the ethmoid cells and sphenoidal sinus are sometimes enlarged after a suppurative inflammation of their mucous membranes. As a result of decomposition of the pus, or by prolonged irrigation with stimulating solutions (carbol, creolin, lysol, etc.), it may proceed to the formation of superficial erosion ulcers on the mucous membrane, and may then be confounded with glanders.

Symptoms.—The following are the morbid indications of catarrh of the sinuses of the upper jaw and head:

1. *Discharge from one nostril:* This is always the first sign observed. At the outset, if the inflammation be intense, this discharge is sometimes mixed with traces of blood. Its consistence is various, as is also its amount. It usually presents a muco-purulent and thickish aspect, and is without special smell; but may also be discoloured and malodorous. When the head is held down, it not unfrequently flows in larger quantity.

2. A *one-sided glandular swelling* in the throat which, by new growth and induration of connective tissue, gradually becomes hard.

3. *Bulging of the frontal* or *superior maxillary sinuses.* This usually somewhat diffuse expansion can be noticed by comparison with the sound side.

4. *Dulness of percussion-sounds* on the diseased side, owing to the collection of catarrhal secretions in the cavity and to thickening of the mucous membrane. We must observe,

however, that when such secretions are only moderate, this dulness may be lacking, so that a negative result of percussion does not disprove the presence of catarrh in the superior maxillary sinus.

The *course* of the complaint is almost always chronic, and may extend over months and years. As rare occurrences, brain symptoms may also appear; when the inflammation spreads to the brain and its membranes (purulent meningitis, cerebral abscess), or if there be a suppurative inflammation of the ethmoid cells and sphenoidal sinus. Horses then present the aspect of sleepy staggers or brain inflammation.

Diagnosis.—Although it is not difficult to recognise catarrh of the sinuses from the indications given, there is yet another method of securing diagnosis, viz., trephining. This operation has a certain importance when it is desired to know if the catarrh be a simple one and not specific, or whether caused by glanders. When the nasal discharge and glandular swelling are on one side only, we are sometimes tempted to suspect glanders. The credit of introducing trephining as a method of differential diagnosis in such cases belongs to Haubner. Whereas, in simple catarrh the mucous membrane is, on trephining, often found to be smooth, in glanders it is frequently lumpy and uneven, and the glanderous process spreads readily to the flaps of skin formed by the operation. But even trephining does not give a positive result in all cases, for in simple, non-glanderous catarrh we not unfrequently perceive small, lumpy protuberances upon the mucous membrane. (See the differential diagnosis of Glanders.)

Sand has observed a mucous degeneration of the nasal conchæ in young horses, which in its morbid aspect closely resembles inflammation of the sinuses. The disease produces great expansion of the hollows of the nasal conchæ, of the upper jaw and of the frontal bone, causing muco-serous accumulations therein, which produce periodical nasal discharges, great distension of the neighbouring bones and difficulty of breathing.

Therapeutics.—A treatment of chronic catarrh of the sinuses is only successful when preceded by trephining; inhalations, as in chronic nasal catarrh, are useless. Trephining renders possible the unhindered outflow of the accumulated secretion, which, by decomposition, continually renews the

inflammation, and also a direct treatment of the affected mucous membrane. It consequently always leads to cure, provided there be no more deeply seated changes, nor any new growths. One may, by trephining, open up the higher division only, but it is better to operate between the two, so as to render both accessible; but occasionally it is advisable to trephine in two places, corresponding to the upper and lower divisions of the cavity. Through the opening made, inject solutions of creolin, lysol, sublimate, carbolic acid, sulphate of zinc, nitrate of silver, etc., and keep the openings unclosed until a complete cure is effected. It may not be out of place to suggest that the injections should be made if possible when the animal is standing, as when lying down some of the secretion may be drawn into the lungs and set up broncho-pneumonia.

New Growths.—Of the new-growths of the adjacent cavities, in addition to the congenital mucous cysts (pseudo-hydrops, spurious dropsy) and dental tumours, the sarcomata and carcinomata of the maxillary sinuses have a practical importance in the horse. The carcinomata develop either from the squamous epithelium of the oral cavity (hard portion of the gums, dental alveoli), and thence extend to the maxillary sinuses, or it is a question of a misplaced germinal bud of squamous epithelium in the bone itself. The sarcomata develop from either the mucous membrane or the periosteum, or they break in from the cavity of the orbit into the maxillary sinus. Both of these tumours are characterised by pronounced elevation of the bones in the neighbourhood of the maxillary sinus, a prominently marked unilateral swelling of the glands of the throat, and at a later stage by a discharge from the nostril of the same side. Sarcomata as well as carcinomata destroy the neighbouring bones; and break into nasal fossæ and also into the oral cavity, which leads to loosening and falling out of the molar teeth. Their treatment by trephining of the maxillary sinus and extirpation of the morbid growth is generally fruitless, as the diagnosis is usually made for the first time when the disease has arrived at an advanced stage.

CHRONIC CATARRH OF THE GUTTURAL-POUCHES IN HORSES.

Etiology.—This is a somewhat rare condition. It usually occurs on one side, rarely on both. A primary affection of the mucous membranes of the guttural pouches scarcely ever happens, but catarrh usually spreads to them from some inflammatory process in the pharyngeal cavity, thus, in pharyngitis and strangles, or in consequence of the penetration of foreign bodies (fodder, fragments of wood, etc.), through the Eustachian tubes. How far congenital enlargement of the pouch is etiologically concerned therewith cannot be determined. Chronic catarrh of the guttural pouch also occurs as

an accompaniment of chronic glanders. Possibly an inflammation of the parotid glands may spread to the pouches. The usual chronic course of the disease arises from the difficulty of removing the exudation gathered in the pouch by way of the Eustachian tubes, and from consequent decomposition.

Anatomical Conditions.—The mucous membrane, which at first is red and swollen, becomes thickened on long continuance, and consequently shows fibrous, nodular infiltrations and a lumpy surface, on which mould-fungi (aspergillus fumigatus, etc.) not unfrequently settle. The secreted matter is either tough and mucous, or more rarely of serous consistence or like pus, when the pus may thicken into caseous lumps. Sometimes, in consequence of the putrescent decomposition, it is ichorous, and the pouch distended by the gases thus generated (tympanites or meteorism of the guttural pouch). The pouch is occasionally found stuffed with fodder, which has entered either through the Eustachian tubes or through an opening made by an ulcer. Finally, and when the process is very much prolonged, we have so-called chondroids in the pouch, *i.e.*, thickened and variously degenerated masses of mucus and pus, of a hard and cartilaginous consistence, with round, often facetted surface, and ranging in size from an acorn to a chestnut or a goose-egg. In one case Johne found over 300 such concretions and Savarese about 250 in the guttural pouch of a horse.

Symptoms.—Chronic catarrh of the guttural pouch in horses manifests itself as follows :

1. By a one-sided (more rarely bi-lateral) nasal discharge of mucous, muco-purulent, purulent and lumpy character, which is sometimes also bad in colour and smell, and when the head is sunk (in grazing, coughing) is frequently emitted in sudden rushes.

2. By swelling of the glands in the cavity between the branches of the lower jaw, such swelling being mostly on one side only.

3. By a swelling in the region of the parotid gland, which varies from fluctuating to doughy when pressed, or perhaps, owing to accumulation of air, is elastic and puffy. Sometimes on pressure it grows smaller, when the discharge is at once increased ; and if charged with air (meteorism of the pouch) a tympanitic tone is on percussion emitted.

4. By difficulty of swallowing, a rattling respiration and

even risk of suffocation when the pouch is much distended. In one case Dobesch observed hæmorrhage from the catarrhally affected pouch which led to asphyxia. Sometimes, too, there is a lateral deviation of the position of the head towards the sound side. In very rare cases, a muco-purulent discharge from the ear has been observed, in consequence of the perforation of the drum. The *course* of the disease is chronic, and may last for years, even for the animal's entire life.

Diagnosis.—This is difficult in chronic cases, and can only be made with certainty when a visible swelling of the pouch can be detected externally. Its similarity to the aspect of glanders is of the utmost importance, because the one-sided chronic discharge and one-sided glandular swelling both occur in the latter, so that for this reason the greatest caution is necessary. Still more easily can chronic catarrh of the pouch be mistaken for a simple chronic nasal catarrh, because the latter frequently accompanies the former; the same also with chronic catarrh of the sinuses, because in these sudden gushes of discharge through the nose are observed. Finally, swelling and abscess-formation in the sub-parotid lymph-glands (upper cervical glands) may lead to the belief that we have a chronic catarrh of the pouch before us. The proposal to ensure diagnosis by introducing Günther's catheter (through the nasal cavity and Eustachian tubes) is not always easy to follow, nor always without danger. Thus Haubner saw the operation several times result in pneumonia (due to foreign bodies), owing to the secretion having been inhaled into the lungs.

Therapeutics.—The treatment of chronic catarrh of the guttural pouch by internal remedies or inhalation is useless. It can only be cured by operation, *i.e.*, by surgical opening and local application of astringent and disinfecting fluids. Respecting the method of such operation (hyovertebrotomy) consult the text-books on operative science and surgery.

Tympanites of the Guttural Pouch.—In addition to the tympanites which arises in the course of catarrh of the pouch from the development of gases of decomposition, there is also an abnormal accumulation of air resulting from the penetration of atmospheric air through the Eustachian tubes during the act of swallowing. We find this form of tympanites especially in foals, and it is attributed to anomalies of construction in these tubes which render possible the passage of air. Sometimes these air-accumulations cause much difficulty in breathing. The treatment is a surgical one (section of the pouch).

ŒSTRUS LARVÆ IN SHEEP.

GAD-FLY VERTIGO. SWINGING SICKNESS.

Definition.—The disease caused in sheep by œstrus larvæ arises from the presence of the larvæ of the sheep gad-fly, œstrus ovis, in the cavities of the nose and sinuses of the head, as well as in the hollow of the horns. This is revealed partly by symptoms of a chronic catarrh of one or all of the cavities named and partly by certain brain disturbances.

Natural History Notes.—The gad-fly of sheep is a small, yellowish-grey and almost naked fly, about ⅓ inch in length and very common in Germany, which, particularly in hot and dry summers, settles in the crevices of sheep-folds, near where sheep pasture, on the edge of woods and in low bushes, and swarms in the hot noon-hours from July to September. The impregnated females then seek a flock of sheep, but on the approach of the flies the sheep become restless, run away or hold their heads near to the ground, thrust their noses between their fore feet, or stand close together in a circle with heads depressed. Immediately after the larvæ have been deposited near their nostrils, the sheep shake their heads, run to and fro and rub their noses against hard objects or on their fore feet. But these signs of irritation soon pass away and for nine months the animals seem to be perfectly healthy.

The larvæ, which are probably already in that stage when deposited, creep into the cavities of the nose and forehead, also into sinuses and the core of the horn, where after about nine months they reach maturity. Whereas at first they are very small and as fine as hairs, they attain a length when ripe of from ¾ to 1¼ inch, have in front two large, claw-shaped mouth-hooks, are yellowish-brown in colour, with darker transverse bands on the eleven rings which form the body. The mature larvæ emigrate in spring, from March to May (seldom earlier or later), and it is during this emigration that they produce the irritation of the mucous membranes of the nasal and accessory cavities and also of the brain known as œstrus sickness. In twenty-four hours from their emigration they change to the chrysalis state, and six to seven weeks later the perfect insect flies away.

in length (the female from 3 to 5 in. and the male from $\frac{3}{4}$ to $1\frac{1}{4}$ in.), is lancet-shaped, white or yellowish-white in colour, broad in front, pointed behind, and is frequently rolled up, having about ninety segments or rings, so that it resembles a tape-worm in appearance. The rounded head has a round mouth-opening on its lower side encircled by a chitinous ring, and near it four slit-like openings for its four legs, each of two members and furnished with a terminal claw, which legs can be drawn in at will. These slits were formerly taken for mouths, hence the name pentastoma or pentastomum, "having five holes."

2. *Pentastoma denticulatum* (linguatula denticula), the larval form of the above, is found most frequently in sheep, also in oxen, hares, rabbits, deer, dromedaries, etc., and likewise in man. It occurs encysted in the liver, mesenteric glands, kidneys and lungs, and also free in the abdominal cavity and in the breathing passages. The larvæ are flat and elongated, white and transparent, about $\frac{3}{16}$ inch long, and divided into some eighty segments, which are furnished with numerous spines (hence the name "denticulatum"). Their four or six feet are armed with double claws, and the sexual organs are rudimentary.

The *process of development* of pentastoma tænioides is as follows: The females living in the nasal cavities of carnivora lay about half a million eggs each, which issuing thence along with the nasal mucus upon the pastures, are there taken up by the grazing animals. The exterior husk of these eggs is dissolved in their stomachs, setting free a mite-like embryo, $\frac{1}{25}$ inch long, with from four to six legs and provided with a tail. This embryo pierces the wall of the stomach, and makes its way to the parenchymatous organs, such as the mesenteric glands, liver, kidneys, etc., and there becomes encysted. In cattle the parasite occurs mostly in the mesenteric glands, less frequently in those of the pelvic and lumbar regions, but also in the liver and spleen (Ostertag). According to Babes, this pentastomum is in Roumania found incidentally in almost all oxen, and invariably in the mesenteric glands. During half a year the embryo undergoes several transformations and changes of skin, until it has attained the true larval form, when it quits the cyst and passes out into the abdominal cavity. Thence it works its way outwards through the lungs and bronchi, thus sometimes causing death from traumatic œdema of the lungs (Gerlach), or else is taken into the bodies of carnivora with the flesh of the slaughtered hosts. According to Gerlach the pentastoma larvæ which pass out

through the air-channels are very resistant, and may even dry up to a certain point without perishing. They reach the nasal cavities of carnivora either directly through the nostrils or from the pharyngeal cavity. After existing there for four to five months they become sexually ripe, and the pentastoma denticulatum has changed into pentastoma tænioides.

Anatomical Conditions.—The changes wrought in the mucous membranes of the nasal and frontal cavities consist in swelling, redness, inflammation and even gangrene. The secreted matter is muco-purulent and sometimes bloody. It is remarkable that the pentastoma seems to be common in some districts and rare in others. Thus in Berlin we have often found it, and only exceptionally in Munich. Hering only once met with the parasite in Stuttgart, while Colin found it in 64 Parisian dogs out of 630 examined, and from one to eleven specimens in each. Tempel found it present in three cases out of 326 dogs examined in Chemnitz.

Statistics.—Researches made in the Hospital for Dogs of the Berlin School have shown that in a great number of the dogs there (in 13 of 200 post-mortem examinations that is 6·5 per cent.) the pentastoma tænioides is met with somewhat more frequently. The parasite was, however, only present in the larger breeds of dogs, mastiffs and butchers' dogs. It was most frequently met with singly in the ethmoid cells, where it was firmly fixed by its mouth to the mucous membrane and lamellæ of the bone. Owing to the depth to which it penetrates, these cells and its similarity of colour to the nasal mucus, the pentastoma may be easily overlooked, but it can be best recognised by the narrow, bright yellow longitudinal streak of its intestinal canal. In most cases the pathological changes wrought in the nasal cavity were not serious. They consisted in swelling, epithelial desquamation, redness and mucous disintegration of the mucous membrane. In one case there was also hæmorrhagic inflammation of the ethmoid cells. In a dog which had suffered a month from chronic nasal discharge the presence in the nasal mucus of pentastomum eggs could be demonstrated.

Symptoms.—The parasites may exist in the nasal cavities almost without external sign. They frequently produce the symptoms of chronic nasal catarrh. The animals sneeze discharge from the nose, wheeze in breathing, rub their noses on hard objects or against their feet, sometimes sneeze or snort out a few pentastomata, occasionally bleed from the nose and may have difficulty of breathing, and even fits of choking. At times the sense of smell seems to be entirely lost. They do not eat their food from the ground, but only when it is held before them, and are often noticed to grow rapidly thin.

As a peculiar complication in a few cases, perforation of the hard palate has been observed, causing great flow of saliva (Adam, Perdan). Usually the trouble ends after a considerable time in ejection of the pentastoma and in consequent recovery.

In other cases the above signs are complicated with cerebral phenomena, which sometimes cannot be distinguished from hydrophobia. The animals are restless, excited, irritated, snappish, roam about the streets, bite readily, howl loudly and continuously, burrow into their straw, thrust their heads between the bars of their cage and manifest rotary movements, or else seem from the outset to be stupefied and quite apathetic. Occasionally paralysis of the lower jaw has been noticed. In such cases death generally supervenes in a few days. On *post-mortem* we find signs of hyperæmia, also of œdema and slight inflammation in the brain. Of diagnostic importance is the identification of pentastomum eggs in the nasal discharge.

Differential Diagnosis.—More important than any confusion with distemper or a simple nasal catarrh is the distinction between pentastoma sickness and rabies. This is possible only when the parasite can be discovered during life or by a *post-mortem*. It is therefore indispensable, owing to the great similarity of many of their symptoms, to carefully examine the nasal and frontal cavities on dissection, to see if there be any pentastomata, *i.e.*, should there be reason to suspect rabies. We must add in this connection that some observations seem to show (Friedberger, Perdan) that these parasites may cause serious brain disorder and even death without producing any marked changes on the nasal and frontal mucous membranes. The view which has been suggested that in such cases there is an incidental complication of true rabies with pentastoma, cannot always be contradicted with certainty. Such a coincidence must, however, be extremely rare, having regard to the relative infrequency of pentastoma in Germany.

Therapeutics.—A radical cure can only be expected from trephining the nasal and frontal cavities. An opening into the latter can of itself do no harm even if the parasite be lodged in the other cavity, as injections of suitable remedies (creolin benzol, carbolic acid, etc.) can be made from the frontal sinus Mere inhalation of tar-vapour, or the use of sneezing remedies have no practical value.

Pentastoma Denticulatum is said to produce symptoms of peritonitis, inflammation of the bowels, etc., in rabbits (Zürn). Except in the experiments in feeding carried out by Leuckart, no injurious effects have been observed in the herbivora infested by these parasites. Leuckart in his tests found serious destructive changes in the lungs and liver of the hares experimented on, consisting in hæmorrhage, inflammation and greatly ramified, hæmorrhagic passages through the organs named, etc. Ströse found pentastoma denticulatum twice in pigs' livers, and Tempel in a goat's lung. Furthermore, Kulagin has observed that, by feeding the larvæ on calves, the pentastoma denticulatum can, in some cases, be brought in herbivora to full maturity of development. He found pentastoma tænioides in their nasal cavities after an interval of from 5 to 7 weeks. For information on the changes effected by the parasite in the mesenteric glands of cattle, and also concerning the importance of pentastoma from an official meat-inspector's point of view, see Ostertag's work on Meat Inspection.

Pentastomum in Horses.—In a case published by Schwammel, five specimens of pentastoma tænioides were found in the nasal cavity of a horse slaughtered on suspicion of glanders. The animal had a muco-purulent nasal discharge and glandular swellings, frequently snorted and shook its head.

CHAPTER III.

DISEASES OF THE LARYNX.

LARYNGITIS. INFLAMMATION OF THE MUCOUS MEMBRANES OF THE LARYNX.

Classification.—Inflammatory conditions of the laryngeal mucous membranes often exist alone. Sometimes the neighbouring organs also participate, especially the mucous membranes of the pharynx, nasal cavity and trachea. In such less definitely localised cases the term "laryngitis" can only be used when the *symptoms are most pronounced in that organ.* When this is not the case, we should speak rather of "catarrh or inflammation" of the upper air-passages. As in other inflammations of mucous membranes, we have to distinguish various degrees of intensity. Thus we divide laryngitis into catarrhal, phlegmonous, croupous, diphtheritic and ulcerative. In accordance with its causes, we may also define it as traumatic, rheumatic, infective, tuberculous, glandular or sympathetic; and from its course, as acute and chronic. We content ourselves with discussing the three following varieties:

1. Acute catarrh of the larynx.
2. Chronic catarrh of the larynx.
3. Croupous and diphtheritic inflammation of the larynx.

I. ACUTE CATARRH OF THE LARYNX. LARYNGITIS CATARRHALIS ACUTA.

Occurrence.—The acute catarrh of the larynx known ordinarily as a swollen throat or sore throat, is very common in horses and dogs, they being more exposed to injurious external influences than other domestic animals. Among the dogs treated in the Berlin dog-hospital three per cent. are afflicted with laryngitis.

Etiology.—The usual cause of primary independent catarrh of the larynx is cold. The inhalation of cold air, drinking very cold water, or severe chill of the skin by heavy rain, cold winds and draughts, etc., very soon cause catarrhal laryngitis, especially in pampered or delicate animals. Hence the almost epidemic appearance of the complaint in spring or autumn. Traumatic and chemical agencies acting on the mucous membranes of the larynx are further sources of the trouble, also external pressure on the larynx, persistent barking in dogs, irritation caused by foreign objects, drugs, smoke, pungent vapours, especially those of chlorine and sulphur. Also the so-called " mash-cough," which occurs in cattle on being fed for the first time on potato mashes, seems to have a similar origin, as probably certain substances contained therein, especially alcohol, exert an irritating influence upon the laryngeal mucous membrane, to which by degrees the animal accustoms itself.

Acute laryngitis occurs secondarily as an accompaniment of various general diseases, such as glanders, distemper, pleuropneumonia, malignant catarrh, etc. Moreover, a catarrhal process often spreads from the cavities of the nose and pharynx, the trachea and bronchi to the larynx. Information on infectious laryngeal catarrh, appearing epizoötically among horses and cattle, will be found further on in this chapter.

Anatomical Conditions.—The pathological changes consist in swelling, redness and even ecchymosis of the mucous membrane, and in accumulation of a serous, mucous or purulent exudation. According to Bruckmüller, it is mainly the upper part of the larynx which is affected, and the laryngitis may sometimes develop to a suppurative destruction of the mucous glands and superficial ulceration of the epiglottis and vocal chords.

Symptoms.—The chief sign of acute laryngeal catarrh is cough, which is at first dry and rough as well as painful, and is either short and broken or else prolonged. Later, it becomes looser and accompanied by a mucous sputum, which dogs at once swallow again. This cough is easily excited by bringing the animals into the open, by eating and drinking, by rising quickly or leaping, by strong excitement, *e.g.*, when dogs in hospital are visited by their owners. The catarrh also manifests its presence by extreme sensitiveness of the laryngeal region to pressure, which the animals try to evade and which

often produces paroxysms of coughing. Owing to great swelling of the mucous membranes and œdematous infiltration of the crico-arytenoideus posticus, breathing is also impeded, and we may sometimes hear from a distance a rattling, whistling or wheezing sound, which on auscultation of the larynx may be clearly heard with other noises, such as rhonchi. In horses the head is frequently held somewhat stretched out. In addition to these most important signs of catarrh of the larynx, we notice when the condition is prolonged, the symptoms of pharyngitis, bronchitis, or rhinitis, diminished appetite, impeded swallowing, dryness of the mouth, nasal discharge and sometimes slight fever. The latter is always referable to complication with one of the other symptoms named. A higher degree of fever always awakes suspicion of a graver, especially an infectious disease.

Differential Diagnosis.—The chief symptom of catarrhal laryngitis, viz., the cough, occurs not only in the other inflammations of the larynx, but also in a further series of diseases of the breathing apparatus, such as tracheïtis, bronchitis, pneumonia and pleuritis. Diagnosis cannot therefore be based on the cough alone, we must note also the other signs, such as increased sensitiveness of the larynx to pressure and the abnormal laryngeal breathing sounds. Tracheïtis and bronchitis are essentially characterised by the presence of tracheal or bronchial rattling sounds ; pneumonia and pleuritis are diagnosed by physical examination of the lungs or thoracic wall. A differentiation between simple and severe inflammatory processes in the larynx is not always easy. We usually attempt definite diagnosis from the severity of local symptoms, the degree of fever, the gravity of the general malady and, above all, from the nature of any matter which may be coughed up, as well as from certain characteristic breathing sounds.

Therapeutics.—In acute laryngeal catarrh prophylaxis consists in hardening the animals and accustoming them to all degrees of weather. Against the disease itself we recommend rest and avoidance of hard labour, especially in unfavourable weather ; moreover, good ventilation must be provided. The application of moist warmth to the region of the larynx is the general practice, viz., in the form of Priessnitz's compresses, and the results are often good. Inhalation of steam is also often tried. The cough may also be combatted

by internal administration of narcotics, especially morphia and codeïn. For dogs, we prescribe morphin in the following form: ℞. Morph. hydrochlor., 1½–3 grs.; Aq. amygdal. amar.; Aq. destill. āā 5½ drs.; M.D.S.—10 to 15 drops, three times a day. Or the same recipe with this variation: that instead of 5½ drs. of Aqua. destillata, 10 oz. 5 drs. are taken, and then a tablespoonful or a teaspoonful may be given. The prescription for codeïn is: ℞. Codeini phosphorici, 4½ grs.; Sirupi althææ, 154 mins.; Aquæ destillatæ, 5 oz. 2½ drs. Sometimes the codeïn seems to be more effective than morphin.

Epizoötic Laryngo-tracheal Catarrh in Horses.—In the summer of 1888 an epizoötic catarrh of the upper air-passages raged widely throughout all Germany, in which twenty regiments in ten different army-corps in Prussia alone were attacked, but which also occurred in private stables. This broke out again in the succeeding years, especially in 1893, 1895, 1897, and 1899, but not so universally. The disease was remarkably contagious, and not rarely all the horses of a stable or of a division fell ill in a few days. Infection was transmitted by the exhaled air; for horses were smitten through being merely led past a sick animal at a few paces' distance. It also attacked impartially horses which had already survived murrain, pleuro-pneumonia or influenza. The incubation stage only lasted a few days. Its chief symptom was in all cases a dry, strong, sharp cough (seldom faint or painful), as well as great sensitiveness of the larynx and trachea to palpation, which operation at once brought on a violent coughing fit. As the disease advanced, a very slight nasal discharge usually began, at first watery and afterwards mucous, complicated sometimes with a slight swelling of the submaxillary lymphatic glands. Auscultation and percussion of the lungs gave, as a rule, no abnormal result. In some of the outbreaks (1893) conjunctivitis also set in. Fever was not regularly present, but occurred sometimes during the first few days of the malady, commencing then sometimes with shivering fits lasting for hours, but only occasionally did the temperature reach 106° Fahr. Rust says that among 426 sick horses the temperature of 173 exceeded 102° F. Only in the most feverish cases was appetite disturbed, the breathing quickened to twenty or twenty-four, the sensorium dulled or bodily force abated. In the immense majority of cases the course was very benign, the illness not lasting more than from eight to fourteen days, and after about a week of convalescence the horses could return to work. This period rarely lasted longer. Only occasionally, and when the sick animals were put to severe labour or exposed to sharp cold, were complications observed with colic, intestinal catarrh, pneumonia or pleuritis. A lasting immunity did not, as a rule, occur.

The *treatment* was purely dietetic: avoidance of severe bodily exertion, moderate exercise in the open. Disinfecting processes yielded no result. Some observers (Zürn) recommended artificial infection of all the threatened horses as a means of shortening the epidemic. This latter is now regarded as a peculiar and hitherto little known infectious disease, which was formerly very likely included under the name of "influenza in its wider sense." But many of the descriptions have an undeniable

resemblance to murrain (the contagious pleuro-pneumonia of horses) and influenza (the distemper of horses), and a careful examination would be well undertaken to enable us more clearly to discriminate between these various diseases.

Epizoötic Catarrh of the Air-passages in Cattle.—Also in cattle the appearance of an enzoötic and even epizoötic outbreak of catarrhal affections of the air-passages has at times been observed (rhinitis, laryngitis, tracheïtis, bronchitis). Such an outbreak occurred in Bavaria in 1890. The disease was accompanied by high fever and ran a fairly regular course. It was by many connected with the influenza (*la grippe*) then raging among men. Its chief symptoms were: mucous nasal discharge, frequent and painful fits of coughing, increased redness of the nasal mucous membrane, great sensitiveness of the larynx to pressure, dyspnœa, increased vesicular respiration and rattling sounds in the lungs, diminished appetite, high fever (104–107° Fahr.), shiverings, tripping, stumbling, etc. In a few cases there was also conjunctivitis catarrhalis. The severest cases usually lasted only a few days, their total duration being eight days, and occasionally two or three weeks. The phenomena were in almost all animals alike, and so also was the thoroughly typical course. Several animals in one byre always sickened together. In solitary cases the appearance of catarrhal pneumonia was noted as a complication, which was manifested by irregular dulness to percussion and bronchial breathing. In such single instances the complaint led to confusion with pleuro-pneumonia, and also, owing to the catarrhal affection of the tarsal conjunctiva, with malignant catarrhal fever. The course was in general very benign. Owing to the typical process of the disease, treatment was seldom required. As regards the connection between this disease and the influenza of man, fuller proof is lacking, although the possibility of such connection cannot be denied.

2. CHRONIC CATARRH OF THE LARYNX. LARYNGITIS CATARRHALIS CHRONICA.

Etiology.—Chronic catarrh of the larynx, or, as it is commonly called, spasmodic cough, or irritation cough, occurs oftenest in horses and dogs, and is very widespread in spring and autumn. Among dogs, older and long-haired terriers suffer most, also Pomeranians, pugs, poodles and spaniels. Of horses, those used for riding and better-class carriage-work suffer most frequently. The causes are essentially those given for acute catarrh, out of which, in fact, most chronic cases develop. Cold plays the chief part, which accounts for the almost epidemic character of the disease in spring and autumn. Chronic conditions of irritation may also arise in the larynx from traumatic causes, or may remain behind after acute infectious disease; thus in horses after pleuro-pneumonia and strangles, or in dogs after distemper. A further source of chronic laryngitis is found

in tumours arising in the larynx, such as papillomatous proliferations, polypi, sarcomata, carcinomata, actinomycomata, glanderous, tuberculous and cystic new growths which maintain a chronic catarrh of their surroundings. In one case Nocard observed chronic cough, or catarrh of the larynx, in a dog, resulting from pressure of a tumour of the mediastinum upon the vagus.

Whooping Cough in Man.—We cannot share the opinion expressed by Röll and others that the so-called spasmodic or inflammation cough of horses and dogs is identical with the whooping cough (tussis convulsiva) of man, because there are no proofs for this assumption. Whooping cough in man represents a contagious, infective disease, which, according to some authorities, is to be regarded as mycosis of the mucous membranes of the larynx and wind-pipe, and, according to others, as neurosis of the laryngeus superior, which especially attacks children, never recurs when once it has been passed through, and is marked by paroxysmal fits of coughing with intervals of quiescence. All these characteristic signs are absent in the spasmodic cough of animals. It is not contagious, attacks chiefly older animals, is remarkable for its frequent recurrence and has no typical course. The epidemic diffusion of laryngeal catarrh, which is specially observable in spring and autumn, cannot be regarded as a proof of its identity with whooping cough ; for every catarrh may take on an enzoötic character from the general influence of similar causes.

Anatomical Conditions.—The pathological changes in chronic catarrh of the larynx are precisely the same as on other mucous membranes. In consequence of chronic hyperæmia the blood-vessels are permanently dilated, and the mucous membrane is thickened and uneven. According to the locality, papillary protuberances (proliferation of the papillary body), or very small nodular irregularities lie upon its surface, giving it a granulated appearance (enlargement of the mucous glands, laryngitis granulosa) ; these last may even suppurate, and lead to the formation of small ulcers. The epithelium, being cast off more freely, forms white patches upon the membrane ; the connective tissue is hypertrophic and permeated by white blood-corpuscles. But in dogs we have often, very strangely, failed on superficial examination to find any visible changes of the laryngeal mucous membrane.

Symptoms.—The most important and often the only symptom is a cough. This is usually dry, rough and croaking, but is sometimes moist and combined with a humming or rattling sound. Frequently there is at the same time a certain degree of dyspnœa. In horses the cough often occurs in fits, with hard stretching of the head and neck. In immediate con-

nection with the cough in dogs we often notice spasmodic choking, the neck being stretched and the head held close to the ground, and this is usually combined with vomiting. This choking and vomiting are caused by the tough, thick mucus coughed up from the larynx into the pharynx. It is also characteristic that in dogs the coughing fits occur with greater severity by night, which evidently arises from the gradual accumulation of this mucus. The general condition is not disturbed. The animals are otherwise quite lively, free from fever, their appetite is normal, breathing unimpeded, and auscultation or percussion of the lungs yields negative results. The duration of chronic catarrh of the larynx is mostly somewhat long, and extends over weeks, months, and even years. In this respect, and owing to numerous relapses, the prognosis of the disease is very unfavourable.

Therapeutics.—Here great quiet and the avoidance of all excitement are of primary importance, as every attack of coughing only increases the hyperæmia of the laryngeal membranes. We also recommend hydropathic wrappings in the form of Priessnitz's compresses for the throat. Derivatory inunctions of the skin round about the larynx (tartar-emetic ointment) were formerly much used. But the matter of most therapeutic importance is local treatment of the affected membranes. Although not quite such good results are obtainable here as from similar treatment in man, yet this method is by far the best. It consists in the inhalation of vapour (steam, aromatic flowers, creolin, carbolic acid, tar or oil of turpentine ; 1 per cent. solutions of sodium chloride, ammonium chloride, bromide of potassium, tannin or nitrate of silver) ; in insufflating powder into the larynx (morphia, nitrate of silver, with 1½ grain : 15½ grs. of sugar) ; also in painting the mucous membrane with a brush or small sponge. The liquid specially used for this purpose is a 1 to 5 per cent. solution of nitrate of silver. For inhalation a sac-like mask is employed for horses, but in case of necessity this may be replaced by a rug held over the head. The inhaling apparatus used for man may be utilised for dogs also, or large inhalation chests, such as are customary in veterinary hospitals, may serve. For horses Dieckerhoff recommends the direct injection of astringent fluids into the larynx. He uses a bent, hollow needle, which, in horses, he pushes into the crico-tracheal ligament and twists upwards towards the larynx. The fluids used are: a ½ per cent. solution of alum, a ½ per cent. solution of

sugar of lead and a 5 per cent. mixture of subnitrate bismuth. We can to some extent confirm the efficacy of these injections, although their application is not entirely without danger. In dogs we make the injection through the crico-thyroid ligament.

The internal administration of remedies to allay the cough is much less efficacious than direct treatment, but may appropriately be combined therewith, especially for dogs. For them we generally prescribe morphia in conjunction with prussic acid or codeïn. To horses one may give expectorant powders upon their fodder, *e.g.*, ℞ sal-ammoniac 1½ oz.; common salt; pulv. liquorice, āā 3 oz. Sprinkle a tablespoonful on each feed. An addition of bromide of potassium or iodide of potassium increases the efficacy of the latter recipe.

3. CROUPOUS AND DIPHTHERITIC INFLAMMATION OF THE LARYNX.

General Notes.—Croupy laryngitis, which is also known as laryngeal-angina, croup or angina, does not in most cases confine itself to the larynx, but the croupous process usually attacks at the same time the trachea as far as the bifurcation of the bronchi. The inflammation also spreads very often to the pharynx, as happens reversely in pharyngitis. Nevertheless, in laryngeal-angina the larynx is the primary and most important seat of disease, whereas in pharyngeal angina this applies to the mucous membranes of the pharynx.

Occurrence.—Laryngeal croup is commonest in cattle and next to them in horses and cats. Other kinds of animals suffer much more rarely.

Etiology.—This croupy inflammation of the laryngeal membranes seems to arise most frequently from cold. In this respect rapid changes of temperature, especially in mountainous regions, passing the night out of doors, and the consumption of very cold food and drink are chiefly blamed. Forneris states that the ailment is particularly rife in the mountains of Piedmont. Along with cold we must also cite traumatic injuries of the membranes of the larynx as a cause of croup, also the action of foreign bodies, of heated air, smoke, pungent infusions, among which ammonia is particularly dangerous, also of irritant gases, such as vapour of chlorine, or of sulphuric acid, etc. Occasionally, owing to the enzoötic appearance of the malady, it almost seems to be of infectious origin. Diphtheritic

affections of the laryngeal mucous membranes, *i.e.*, necrotic processes in the tissue of the membrane itself, with formation of ulcers, produce sometimes an extreme degree of croupous inflammation. In other cases we meet with it as a secondary phenomenon in the course of certain infectious diseases, such as the malignant catarrh of cattle, petechial fever and acute glanders.

Anatomical Conditions.—The changes wrought by laryngeal croup upon the mucous membranes of the larynx and trachea consist at first in an insular deposit like hoar-frost, which later becomes confluent and gradually thickens to a grey or whitish-yellow (in cattle even yellowish-brown) membranous coating, which often forms perfect casts of the larynx and trachea and is sometimes easy, sometimes difficult to dislodge. The microscope shows this coating to consist mainly of fibrinous threads or stalks, white blood-corpuscles as well as free nuclei. The subjacent membrane is hyperæmic and even hæmorrhagic, being also swollen and infiltrated with pus. The sub-mucosa is frequently in a state of œdema. In diphtheritis of the larynx we often find discoloured membranous deposits, greyish-white to greyish-yellow with a tinge of green, which leave ulcerous losses of substance of the mucous membrane behind them, and sometimes spread forwards in the pharynx and to the root of the tongue, and downwards to the trachea and bronchi. As complications of croupous or diphtheritic laryngitis we must indicate: tracheïtis, bronchitis, croupous pneumonia, the pneumonia due to foreign bodies, pulmonary emphysema.

Symptoms.—Laryngeal croup signalises itself by the rapid and sudden appearance of severe illness. The general condition is from the first gravely compromised, the disease often setting in with very high fever and shivering fits. Then amid violent attacks of coughing much difficulty of breathing develops. The animals breathe more or less dyspnœically with considerable distension of mouth and nostrils, stretch out their heads, slaver profusely, sometimes press forward against the bar, or stand anguish-stricken with outspread forelegs, their mucous membranes being very red, their eyes prominent and glaring and the veins of the skin charged to repletion. The region of the larynx is swollen, hot and sensitive to pressure; indeed, a sharp pressure of either larynx or trachea may cause risk of suffocation. Meanwhile each respiration is accompanied by the most varied

sounds: groaning, whistling, rattling, flapping, buzzing; and on laying the hand over the larynx we can distinctly feel a laryngeal fremitus. If these symptoms rapidly increase, death may follow from asphyxia in a few days. If, on the other hand, the malady takes a milder course, then after from three to five days membranous croup-shreds or tubular casts of the larynx and trachea begin to be coughed up through the mouth and nose, after which recovery quickly follows; and the entire duration of the sickness will not exceed five to six days. Among other accompanying symptoms are: suspension of eating and rumination, salivation and regurgitation, constipation and suppression of milk. Sometimes the animals perish from subsequent inflammation of the lungs. The aspect of laryngeal diphtheritis is similar, though mostly more intense.

Prognosis.—In cattle and horses the prognosis must at best be termed doubtful, as suffocation may occur at any moment. Recovery may, however, come as rapidly as death. A forecast seems to be more favourable in cats, for we have several times seen the complaint with them run a perfectly mild course.

Therapeutics. — The treatment of laryngeal angina is, owing to its very rapid course, somewhat precarious. Usually we apply inhalations of steam or lime-water, Priessnitz' compresses, continuous hot poultices, friction with quicksilver ointment, and also astringent fluids, such as solution of nitrate of silver. Trasbôt recommends bleeding cattle, with subsequent administration of iodide of potassium in daily doses of 2½ drams. The most important point in treatment is that there be no delay in performing tracheotomy if the dyspnœa becomes severe; very often this is done too late. For cats and dogs an emetic may be tried.

ŒDEMA OF THE GLOTTIS. ŒDEMA OF THE LARYNGEAL MUCOUS MEMBRANE.

Definition.—Under the name of œdema of the glottis we describe an œdematous swelling of the sub-mucous connective tissue in the larynx, which sometimes exhibits the characters of a serous or suppurative inflammation (phlegmon), and sometimes that of a true œdema due to obstruction.

Etiology.—1. An *inflammatory* œdema of the glottis either arises primarily in the course of intense laryngeal inflammation

after injury of its mucous membranes by foreign objects or pungent drugs, as well as after cold, *e.g.*, under rapid changes of temperature after a previous laborious march through much dust; or else it arises secondarily as a collateral œdema in connection with the inflammation of some adjoining membrane, such as a severe pharyngitis, also in the course of many infectious diseases, *e.g.*, anthrax, glanders, petechial fever, pox, pyæmia or pneumonia.

2. *True congestive œdema* of the larynx arises from congestion of blood such as follows heart, lung or kidney diseases, or from compression of the jugulars. We have repeatedly seen it in horses after previous strangulation.

Anatomical Conditions.—The œdema generally attacks only the upper portion of the larynx, *i.e.*, the base of the epiglottis, the arytenoid cartilage, epiglottic ligaments and the ventricular ligament. The lower parts, and especially the rima glottidis with its ligaments, are seldom affected, because there the mucous membranes are more firmly attached to their base. The œdematously swollen mucous membrane forms thick, gelatinous, quivering blisters at the spots named, and particularly on the vocal chords, which project into the orifice of the larynx. These blisters or swellings are sometimes pale and at others very red. On being cut they discharge, according to the nature of the swelling, a serous, purulent or gelatinous fluid. Occasionally the adjoining parts of the pharynx, root of the tongue and nasal cavity are found to be œdematous.

Symptoms.—1. The phenomena of *inflammatory* œdema of the glottis appear very rapidly and suddenly. Its aspect, which is closely related to that of croup, consists essentially in extreme difficulty of breathing, leading finally to asphyxia, in which inspiration is more especially impeded. The animals breathe dyspnœically with much panting, groaning, rattling, and often with a whistling sound which can be heard from a distance. They cough, display fear, outbreaks of sweating, injected mucous membranes, protruding eyeballs, and rapidly perish in a few hours if help be not speedily afforded.

2. In *true congestive* œdema the course is slower, and the severe stages occur only after several days or weeks.

Diagnosis.—The frequent confusion of œdema of the glottis with laryngeal croup is easily explained by the marked

similarity of their symptoms. But the diagnosis of the former is assured by its frequent peracute course, and by the fact that no fragments of membrane are coughed up.

Therapeutics.—The only sure remedy in œdema of the glottis, when blisters in the region of the larynx have provéd unavailing, is the performance of tracheotomy with all possible speed. Other manipulations, such as blood-letting, the use of strong purgatives, the administration of ice-pills, the inhalation of or painting with astringents, may be useful in sub-acute cases, but are of no value in those of rapid course.

ROARING. WHISTLING. HEMIPLEGIA LARYNGIS. STRIDOR LARYNGIS. ATROPHY OF THE CRICO-ARYTENOID MUSCLES. LEFT-SIDED PARALYSIS OF THE VOCAL CHORD.

Nature.—Laryngeal stridor (roaring) is principally due to a left-sided paralysis of the vocal chord (hemiplegia laryngis), the result of paralysis of the recurrent laryngeal nerve, with left-sided atrophy of the openers of the glottis (posterior and lateral crico-arytenoideus, and left half of the arytenoideus proprius).

Occurrence.—Laryngeal stridor is a disease peculiar to horses in their earlier years. Beyond its occurrence in horses, paralysis of the recurrent nerve is sometimes met with in cattle, and also in dogs. In Europe, laryngeal whistling is commonest among the higher breeds of England, France, Hanover and Holstein. The English thoroughbred is, of all horses, most subject to it. This lesion is, however, by no means restricted to thoroughbreds, for it occurs similarly in all kinds of horses, especially in the English and Dutch half-breds, and also in those of inferior breed. It is exceedingly rare in India, Egypt, the Cape, Australia, South America, and especially in Arabia (Fleming, Nocard). Stallions and geldings suffer more frequently than mares.

Etiology.—The causes of laryngeal stridor are not known with certainty. In general, the following influences present themselves for consideration.

1. Hereditary tendency must be placed first as a factor. The higher bred horses, and particularly stallions, transmit it to their issue—a fact severely felt in many breeding studs. The transmission of the disease by heredity is most frequently

noticed in the third year, when the horse is put to work ; sometimes, however, it appears in the first or second year ; or even the fourth year. Horses with long, thin necks seem most predisposed to it. The training of the English race-horse is by many brought into etiological relationship with laryngeal stridor, inasmuch as the heart and aorta are greatly developed thereby, and the recurrent nerve is much more exposed to the pulsations of the aorta on account of the poverty of fat which exists at the same time in all parts of the body. Steamy, badly ventilated stables, and the disposition to catarrh thereby produced, are also blamed as indirect causes.

The question has often been asked why in the vast majority of cases the muscular atrophy is on the left side only. According to Günther, out of 100 cases, 96 are left-sided ; Fleming says 99 and Cadiot 95. Frequently the superficial course of the left recurrent laryngeal nerve in the neck has been blamed. More important for the settlement of this question seems to be its course in the thoracic cavity, to which Günther first called attention. The left recurrent laryngeal nerve covers a greater distance in the thorax, because it winds round the aortal arch. Günther found that in the course of frequent chest diseases of horses (pleuro-pneumonia), the left laryngeal nerve is also affected and produces a left-sided paralysis of the larynx. Franck held that the nerve is overstrained by the aorta, which is so highly developed in thoroughbreds, and to this opinion Martin also adhered. Martin points especially to the embryological side of the question. He believes that the recurrent laryngeal nerve is irritated by the arterial arches, the alternating motion of the heart and the great length of the neck in thoroughbred horses. The constricted position of the recurrent between the aorta and the trachea is also etiologically blamed (Vaerst). According to Sussdorf, the nerve in question is always visibly flattened at the spot where it passes between the aorta and the wind-pipe.

All these views have been overturned by the recent microscopic researches of Thomassen, by which it has been demonstrated that in 99 per cent. of all cases of laryngeal stridor of nervous origin, the changes in the recurrent laryngeal nerve are to be found in its peripheral extremities, namely, in the vicinity of the larynx. The thoracic portion of the nerve, and especially the loop which bends around the aorta, are always found in a normal condition. So that, according to Thomassen, we have to deal with a nervous lesion of a purely peripheral

type. The anatomical changes in the nerves themselves are not, apparently, to be regarded as those of neuritis; but merely as secondary processes of degeneration, with subsequent proliferation of the interstitial connective tissue. The anatomical changes in the nerves in laryngeal stridor are also in every instance further advanced than those in the corresponding muscles.

2. Another cause of roaring is also murrain or pleuropneumonia, from which it remains behind as a sequel. Whether the development of roaring in such cases is connected with an inflammatory affection of the recurrent nerve, which has spread from the inflamed pleura to the nerves, or with some other disturbance of the nervous system (paralysis of the nerves by the action of poison), cannot at present be decided. Horses may become affected with this secondary form of laryngeal stridor at any time of life; but it occurs as a rule in the fifth and sixth years.

3. In the course of serious phlegmonous inflammation of the pharynx and larynx (strangles, angina) a muscular inflammation and muscular atrophy of the larynx occurs, with stenosis of the rima glottidis, in which either a paralysis of the muscles is developed from the very first, or else, later on, a chronic sclerotic inflammation of the connective tissue of the submucosa, with stenosis of the space between the two arytenoid cartilages.

4. Poison may also cause laryngeal whistling, owing to nervous or muscular paralysis. In this connection we must mention chronic lead-poisoning, the eating of chick-peas, mutters, tares and lucerne, which perhaps effect this curious result owing to the presence of certain fungi. According to Fleming, from 2 to 4 per cent. of all cases of this malady are traceable to these vegetable poisons.

5. Finally, we sometimes also observe that compression and paralysis of the recurrent laryngeal nerve in its course along the neck are affected by tumours, *e.g.*, by lymphomata, struma, abscesses of the lymph-glands during strangles, dilatation of the pharynx, and also in the thoracic cavity by enlarged bronchial glands. It has been experimentally shown that accidental injury of the recurrens at this spot produces the same effect. Glockner has described a case of obliteration of the left carotid artery, with consecutive roaring.

K. Günther indicated as true causes of laryngeal stridor: farcy, angina, influenza, lead-poisoning, poisoning with lucerne, lathyrus (chicken

vetch), chick-pea, arachis hypogœa (earth-nut), cold, hereditary tendency. He mentions the following as wrongly attributed causes of it: long, slender neck; pressure-paralysis produced by enlarged glands or the air-tube; pronounced development of the vessels, with poverty of fat; greater length of the left recurrent nerve; and myopathic lesion. He is also against the generally accepted view that paralysis of the recurrent nerve does not require four weeks for its development, holding that, like every peripheral nerve-palsy, it may occur quite suddenly.

According to Malkmus, bilateral paralysis of the vocal chords of an acute nature must be distinguished from the special laryngeal stridor (chronic unilateral paralysis). It is characterised, in contrast to the latter, by extreme difficulty of breathing, suddenly appearing in a condition of rest, or of slight stimulation, as well as by marked difference in the laryngoscopic appearances. It also sometimes causes sudden death. The cases of sudden development of laryngeal stridor, which have been observed by Albrecht, Lies, Vollers and others are curable; but rarely and under special circumstances. Bilateral paralysis is apparently produced by the general effects of chemical agents. According to Voshage, from the statistics of twenty years of cases of laryngeal stridor in the clinic of Hanover, it occurs more frequently in males than in females (12 as against 8 to 9).

Huth traces back the occurrence of paralysis on the left side to mechanical injuries in castration (operation being carried out in the left side position; prevalence of the affection in geldings).

Nocard doubts the heredity of laryngeal stridor and regards strangles as the most frequent cause; on the other hand, Sanson and Labat have advocated the hereditary view.

Hoffmann locates the position of laryngeal stridor, not in the larynx, but in the anterior part of the nasal cavity.

Anatomical Conditions.—The muscles on the left side of the larynx which expand the rima glottidis are atrophied and fattily-degenerated, and are noticeable for their pale, yellowish-red colour and their similarity to stripes of connective tissue (prominence of the perimysium). The atrophy is usually, however, not complete, but affects mainly the posterior crico-arytenoid muscles. This muscular atrophy corresponds almost always exactly with the area over which the recurrent nerve extends. This nerve itself is also often found atrophied and yellowish (fatty degeneration). In rare cases the atrophy is on the right side; but when there has been previous severe laryngitis, it is mostly total. In contrast to atrophy of the other laryngeal muscles, the crico-thyroid muscle is in roaring frequently found to be much developed by way of compensation. The direct consequence of paralysis of the muscles of the larynx is an obliquity of the left arytenoid muscle, which sinks downward, and thus narrows the entrance to the larynx. The lungs are sometimes found in a condition of emphysema.

Symptoms.—The main symptom in paralysis of the recurrent nerve consists in an *inspiratory* sound originating in the larynx, which arises from the arytenoid cartilage of the affected side sinking towards the interior of the laryngeal cavity, in consequence of which the rima glottidis can no longer be sufficiently opened, owing to paralysis of the left vocal chord. This breathing sound (stridor) can seldom be heard when the animal is at rest, as in eating (these are extreme cases), but becomes audible on movement, and is characterised sometimes as whistling or piping, at others as rattling and snoring, snorting, panting and wheezing, or, again, as croaking, screeching, bellowing or roaring. The sound is often so intense as to be heard a long way off. It is usually emitted only during inspiration, and in severe cases during expiration as well. The nostrils are then always widely dilated like a trumpet. The noise is almost invariably loudest after severe exertion, and especially after galloping, but dies away of itself after a few minutes' rest. It is characteristic that the stridor may be momentarily silenced by compressing the nostrils (which are always widely dilated), so that only a small stream of air can enter, corresponding to the narrower rima glottidis. In mild cases the defect can be observed only after forced exertion. As the breathing sounds grow in intensity, so also does the animal's difficulty of breathing, which may at times amount to choking fits and collapse. The buzzing or quivering cough is also remarkable, as during the inspiration which follows each violent expiration, the relaxed left vocal chord vibrates or quivers. In the same way in roarers the neigh is sometimes hoarse.

A further guide in the proof of muscular atrophy may be found in palpation of the larynx. In advanced stages of the complaint the left arytenoid cartilage can be pressed in more deeply than the right when the larynx is grasped by the fingers ; even when at rest, the characteristic laryngeal sounds may be thus set up. According to Bassi, on examining the interior of the larynx, after introducing a gag, we may observe a displacement of one arytenoid cartilage as well as relaxation and obliquity of the vocal chord. This attempt does not, however, always succeed, and it is more important to try an endoscopic examination of the inside of the larynx, and especially of the vocal chords, by Polansky and Schindelka's laryngoscope, a very valuable instrument for this and other purposes.

The *course* of the condition is generally chronic, and, indeed,

it usually tends to grow worse. Roaring develops frequently in the run of acute diseases, especially of pleuro-pneumonia. Cure can only be effected when there is paresis of the laryngeal muscles (partial paralysis), never when the paralysis is complete. Recovery is most probable when the muscular paralysis is a sequel of pleuro-pneumonia or severe laryngitis, and in the toxic form may be secured by avoidance of the offending fodder. The nature of the service required from the sick animals has also great influence upon the course of the condition.

Differential Diagnosis.—Left-sided paralysis of the vocal chords may be confused with roaring caused by other mechanical contractions of the air-passages, such, for instance, as occur in the course of stenosis of the nostrils, posterior nares, pharyngeal cavity and larynx ; or of the trachea as a result of new growths, polypi, cysts, congenital deformities, impressions, fractures, swelling of the guttural-pouch, operations (tracheotomy), etc. In all such cases palpation of the larynx will decide the question, or, if still doubtful, an examination by the laryngoscope. Roaring must also not be confused with other sounds which are made by horses more in the way of play (grunting, snorting, wheezing, blowing, etc.), or by the persons in charge of them.

Therapeutics.—The treatment of roaring and whistling is essentially operative, tracheotomy being most frequently practised, with the introduction of a permanent tube. This procedure does not of itself cure the disease ; but it removes the difficulty of breathing, and makes the horse useful again. Even race-horses may, by tracheotomy, be rendered again capable of racing. Günther has, without satisfactory result, excised the paralysed vocal chord and the sunken arytenoid cartilage after previously opening the laryngeal cavity, and Stockfleth, Fleming and Bassi have followed his method. Möller operates for one-sided paralysis of the laryngeal vocal chord by cutting away the entire arytenoid cartilage. The various laryngeal operations which have been recommended in the treatment of roaring, have not proved satisfactory (arytænectomy). This operation has found but little support, and, from its doubtful value, is now seldom undertaken.

Whether a prolonged course of arsenic or iodine (iodide of potassium), or the application of electricity can induce improvement or cure, as many affirm, must be more thoroughly investi-

gated. In any case such results can be expected only at the beginning of the condition. It is advisable at the outset to try subcutaneous strychnine injections in the vicinity of the larynx ($\frac{3}{4}$ to $1\frac{1}{2}$ grains of strychnine nitrate per diem); omitting the injection every third day. If it be desirable to apply the injections intra-tracheally, the dose must be smaller, say, half the above, otherwise signs of poisoning and fatal strychnine tetanus may occur. Veratrine acts similarly to strychnine. As a prophylactic it is absolutely necessary to exclude from breeding all horses with the hereditary taint. (The laryngeal stridor which follows pleuro-pneumonia, strangles, etc., is, on the other hand, not hereditary).

Forensic Notes.—According to the German Civil law, every case is to be regarded as one of laryngeal stridor "which by a chronic and incurable diseased condition of the larynx, or the air-tube, causes a difficulty in breathing recognisable by an audible sound." The most important and frequent of those disease processes is paralysis of the left vocal chord. In addition to this there are other morbid conditions of the larynx which cause roaring; especially new growths (polypi, cysts) on the epiglottis and the inner surface of the larynx, chronic constrictive laryngitis and perilaryngitis, necrosis of the cricoid cartilage, ossification of the arytænoid cartilages, etc. Laryngeal stridor is also produced, in some cases, by chronic diseases of the trachea, when these are accompanied by a narrowing of the air-passages (new growths: *e.g.*, enchondroma, stenosis, flattening, spiral twisting). The period of guarantee against laryngeal stridor is 14 days, according to the German Civil law.

Spasm of the Glottis (*spasmus glottidis*), which is the contrary of laryngeal paralysis, is rarely seen in our domestic animals. It consists in spasmodic closing of the rima glottidis with extreme inspiratory dyspnœa caused by affection of the superior laryngeal nerve. Little is known as to its origin. Gerlach describes "spasmodic tightness of breath" as a special form of roaring or whistling; but the poisonous agents which he regarded as causing it, viz., chick-peas, etc., are among those which produce paralysis of the larynx. Degive and Ebinger have each described a case of true laryngeal spasms in horses. As regards treatment, injections of morphia or chloral hydrate are recommended.

Syngamus Laryngeus.—According to Railliet, syngamus laryngeus, a variety of strongylus, is frequently met with in cattle in the southern parts of Annam. It is found even in the larynx, in the mucous membrane of the vocal chords and of the laryngeal sinuses, without causing any trouble to the animals.

CHAPTER IV.

DISEASES OF THE TRACHEA AND BRONCHI.

ACUTE BRONCHIAL CATARRH. BRONCHITIS CATARRHALIS ACUTA.

Forms of Bronchitis.—As with laryngitis, so also in bronchitis we may distinguish various forms according to their duration, intensity and locality. There is also an acute and a chronic bronchitis, a catarrhal and a croupous, a serous, mucous, purulent and fœtid. When there is much serous exudation, we speak of bronchorrhœa serosa, or, if specially purulent, of broncho-blennorrhœa. Much importance is also attached to inflammation of the smallest bronchi, the bronchioli, to which the name of bronchiolitis, or bronchitis capillaris, is given. A very rare form is bronchitis villosa. Bronchitis verminosa and mycotica are peculiarly etiological forms. In addition we have infectious, glanderous and tuberculous bronchitis.

Etiology.—Acute bronchial catarrh is commonest in horses, and next to them in dogs and cattle. The disease is seldom con fined exclusively to the mucous membranes of the bronchi, but is often found in those of the trachea and larynx, even the nasal cavity being frequently affected. In these latter cases we speak in general of a "catarrh of the air-passages." The causes are numerous.

1. Among *predisposing agencies* are youth, feeble constitution, poor condition of nourishment, pampering and accustoming animals to warm stables.

2. *Cold* is here a main source of the evil, as in laryngitis, especially the action of cold air in breathing. Damp, wet-cold weather, rapid alternations of season, moist, cold winds and thick mist are very prejudicial in this respect, which explains the frequency of the complaint in spring and autumn.

3. *Mechanical and chemical irritants*, *e.g.*, the inhalation of pungent gases and vapours, hot air, smoke or of air defiled with fungi, as in fodder which is dusty, mouldy or infested with

uridineæ or ustilagineæ, or the penetration of drugs from carelessly administered drenches. Whether inhalation of pollen from flowering grasses produces bronchitis, as occurs in the hay-fever of man, is uncertain. Respecting parasites as causes of bronchial catarrh, see the paragraph on bronchitis verminosa (further on in this chapter).

4. The disease not unfrequently arises from the *spread* of catarrh from the upper air-passages to the lower.

5. In a few cases we must presume the action of *infectious matter*, when, for instance, it is known that cold has not been the cause, or when the malady appears suddenly in several animals at once, with high fever and displaying a strikingly enzoötic and infectious character. Such outbreaks of bronchitis occur sometimes epidemically among horses and cattle, and have been described as "epizoötic catarrh of the air-passages."

6. Finally, bronchitis occurs secondarily as an *accompanying symptom* of all kinds of *infectious diseases*, in which it sometimes represents the primary local affection, thus in the distemper of dogs, pleuro-pneumonia, influenza, glanders, anthrax and pox, or variolæ. For this reason it is not always easy to distinguish a secondary infective bronchitis from a primary, as both may be marked by high fever. It is always well, when suspecting a primary and very feverish bronchitis, to think also of the possibility of a secondary affection in the course of some infectious disease, since an inflammation of the lungs, for instance, may at the outset present the symptoms of bronchitis. The former designation of "catarrhal fever" always includes such a supposition.

Anatomical Conditions.—The most important changes on the mucous membrane are redness, swelling and ecchymoses, the deposit of a serous, mucous or purulent secretion caused by transudation of blood-serum, mucous desquamation of epithelium with breaking down of the mucous glands and emigration of white blood-corpuscles, as well as small cellular infiltration of the mucosa or sub-mucosa. Should the inflammation spread from the mucous membrane to the alveoli, then the parenchyma of the lungs is also affected, and we have a catarrhal inflammation of the lungs (broncho-pneumonia).

Symptoms.—Acute bronchial catarrh begins sometimes with moderately high fever (103 to 107° Fahr.). In seventeen such cases among horses we found fifteen times the maximum degree of fever on the first day, the temperature being in four

cases over 105.8°, in seven above 104°, and in four exceeding 103° Fahr. The pulse also is quickened, and not unfrequently shivering fits, with great lassitude, are noticed. Appetite, rumination and milk-secretion are all suppressed. The cough, one of the most important symptoms in acute bronchitis, is at first very dry and painful, but afterwards becomes looser and moister, and is at last accompanied by a watery, glairy, mucous or even purulent discharge. Upon auscultation of the lungs we hear at first rough, intensified vesicular breathing. Later, this becomes indefinite, and we have dry, rattling noises (whistling, wheezing, buzzing sounds), which grow more moist as the secretion increases. In capillary bronchitis (bronchiolitis) we perceive rustling sounds, especially in dogs. These noises are the characteristic symptoms of bronchitis. In contrast to these serious changes as revealed to the ear, the results of percussion are perfectly normal, so long as there is no development of catarrhal pneumonia. The tightness of breathing is very severe, especially in bronchiolitis, and may even lead to suffocation. Such cases of capillary bronchitis occur mainly in very young or very old animals, in the latter of which the bronchiolitis may speedily be complicated with œdema of the lungs. According to St. Cyr, the danger in bronchiolitis is in inverse proportion to the girth of sick dogs. The *duration* of acute bronchial catarrh is frequently only from four to eight days, but averages two to three weeks. With the exception of very young or very aged animals, its *course* is usually favourable, and the illness ends in complete recovery. In severe cases, and with continuance of its causes, the acute bronchitis may become chronic.

Therapeutics.—In the first place local treatment may be attempted by inhalations of steam, alone or in conjunction with common salt, chloride of ammonium, creolin, carbolic acid, alum, tannin or oil of turpentine. Among internal remedies apomorphin hydrochloride is one of the most valuable expectorants. It is administered to dogs in daily doses of $\frac{1}{14}$ to $\frac{1}{7}$ grain, in 1½ oz. of water, a tablespoonful every three hours. Chloride of ammonium is particularly good for a dry cough, in advanced stages of the catarrh, as well as in slight fever. To horses give it in doses of 2 to 4 drams, and to dogs of 3 to 15 grains. Tartar emetic is most suitable in higher fever or for more robust animals and this also applies to yellow sulphide of antimony, a remedy formerly much used (horses 30 to 75 grains ; dogs ¾ to 3 grains). Chloride of ammonium and tartar emetic may be prescribed

together as a so-called expectorant mixture, *e.g.*, for a dog: ammonium chloride and liquorice juice, āā 1½ drams; tartar emetic, 8 grains; water, 10 oz.; one tablespoonful three times daily (teaspoonful for a small dog). For excessive secretion of the bronchial mucous membranes administer oil of turpentine or terebene (to dogs internally 1½ to 8 minims), senega-root, aromatic spirit of ammonia, etc. The two latter may be prescribed together thus: infusion of senega, 2½ drams to 5 oz.; aromatic spirit of ammonia, 1½ drams. From two teaspoonfuls to two tablespoonfuls daily for a dog. In very painful attacks of coughing we prescribe, as in acute laryngeal catarrh, morphin and bitter-almond water (1½ grains to 2½ drams in 5 oz. of water; daily two to three table or teaspoonfuls for a dog), or codeïn (3 grains in 5 oz.). Should œdema of the lungs threaten, administer stimulants (camphor, ether, caffeïn, hyoscin, alcohol), or give an emetic. In addition to this medical treatment, there must also be careful supervision of the diet, and other hygienic details.

CHRONIC BRONCHIAL CATARRH. BRONCHITIS CATARRHALIS CHRONICA.

Etiology.—There is a natural pre-disposition to chronic bronchial catarrh in all feeble and pampered animals, especially in young dogs and cachectic sheep. Among its direct causes must first be cited those given for acute bronchitis, for the chronic form usually develops from the acute. Then there are certain chronic heart and lung diseases from which it generates secondarily. Among such are valvular defects, chronic interstitial inflammatory processes in the lungs, emphysema of the lungs, all of which cause a regurgitation of blood into the bronchial veins and arteries (the capillary network of the bronchial arteries also communicates with that of the pulmonary artery). On this account most broken-winded horses also suffer from chronic bronchial catarrh. Many general diseases are accompanied by chronic bronchitis, particularly the so-called constitutional diseases, like anæmia, leucæmia, chlorosis, and also chronic Bright's disease. For parasites as a cause of this complaint, see further on in this chapter under the heading of "Verminous Bronchitis."

Anatomical Conditions.—In chronic catarrh the bronchial mucous membrane is a dark-brownish red, traversed

by greatly dilated vessels. It is thickened and sometimes covered with warty excrescences, the secretions are mucous or purulent, often even putrescent and malodorous (bronchitis fœtida), and frequently quite fill the small bronchi, so that the affected lobe of the lung is in a condition of atelectasis, which during further course of the disease is complicated with chronic, catarrhal and interstitial processes in the lungs. When the inflammation is long continued, the bronchial walls become thickened in every part (mucous membrane, the true bronchial tube and the peribronchial tissue) by new growths of connective tissue after previous small-cellular infiltration : endo-bronchitis, meso-bronchitis and peri-bronchitis chronica. A special form of peribronchitis in horses has been described by Dieckerhoff as peribronchitis nodosa. This appears as multiple, tough nodules, greyish-white in section and varying in size from a groat or millet-seed up to a pea; the interior is often filled with caseous and calcified bronchial secretion so that the nodules greatly resemble the tubercles of glanders. They are, however, distinguishable from the latter by their regular quality and equality of age, and by the fact that their centre can be peeled or decorticated—a fact denied, however, by Nocard. According to Liénaux, the peri-bronchitis of horses is of verminous origin; for a næmatode larve is nearly always to be found within each of the nodules. According to Ziegler, peri-bronchitis nodosa should be reckoned as a pneumonia, so far as it concerns the breathing bronchioli, and be designated as nodular bronchopneumonic induration.

One of the most frequent sequels of chronic bronchial catarrh is bronchiectasis, *i.e.*, dilatation of the bronchial walls which, being catarrhally inflamed, are therefore less resistant; this occurs especially in horses at the apex of the lungs. We distinguish a cylindrical, a sacculated and a spindle-shaped bronchiectasis. The bronchiectatic cavity is fully charged with decomposed, thickened mucus and pus, which may even become calcified (so-called "lung-stones," or pulmonary concretions) and attain the size of a pea or chestnut, or even that of the fist. Smaller bronchi are often distended to the thickness of a finger. Finally, another not infrequent accompaniment of chronic bronchial catarrh is emphysema.

Symptoms.—These are in general the same as those of acute bronchitis, of which also the cough is the chief symptom,

and usually combined with profuse secretion of mucus. Auscultation reveals various rattling noises, partly dry and partly moist. The difficulty of breathing is extremely acute whenever the chronic bronchial catarrh is complicated with atelectasis and emphysema. In the horse we then have the phenomena of broken wind. As the disease progresses the animal's condition begins to suffer; it grows thin, feeble and cachectic and sometimes perishes of catarrhal pneumonia. The presence of bronchiectasis is known by the enormous and sudden discharge of sputum, which is usually much decomposed and smells very badly, and also—though but rarely—by a localised, clear tympanitic tone on percussion of the thoracic wall.

Therapeutics.—A cure of chronic bronchial catarrh is often possible, but takes a long time. The remedies are the same as those advised for the acute form. Expectorants, which may be administered both internally and also externally as inhalations, are most frequently used, such as oil of turpentine, tar, creolin, chloride of ammonium, etc. Also the neutral salts, apomorphin, ipecacuanha, senega and quillaja are employed. The intratracheal injections recommended by Levi and others have been already tried by Lafosse 50 years since, but their efficacy is doubtful. In man the treatment of bronchiectasis is partly surgical.

Physostigmine and pilocarpine have latterly been recommended for chronic bronchitis. But we must counsel caution in their use, for in one case, after subcutaneous injection of 1½ grains of the former for a powerful horse suffering from chronic bronchial catarrh, we observed extremely difficult breathing, followed after three-quarters of an hour by death from suffocation as a result of œdema of the lungs. In a similar manner we have seen dogs die from œdema of the lungs after being treated for the same disease with ¾ grain of pilocarpine.

BRONCHIAL CROUP. BRONCHITIS CROUPOSA.

Etiology.—This arises from the same causes as laryngeal croup. Extreme chills, sudden changes of temperature and of weather, the inhalation of smoke in conflagrations or in forges, and the introduction of pungent medicaments into the air-tubes are the usual causes. But in cattle there seems to be in addition a specific, infectious bronchial croup. We also meet with the disease as a co-symptom of croupous inflammation of the lungs, also in many infectious diseases, *e.g.*, in rinderpest, malignant catarrhal fever, and bovine pleuro-pneumonia.

Anatomical Conditions.—The mucous membranes of the trachea and bronchi are injected and swollen, showing yellowish membranous coatings up to $\frac{3}{16}$ inch thick, which possess a smooth upper surface and adhere only loosely to the mucous membrane. Its microscopical constitution is the same as in laryngeal croup. Sometimes we also find rolled-up, ribbon-like masses, especially on the posterior wall of the wind-pipe. Moreover, the finer bronchi are often completely choked with yellow, tough cylinders like thrombi. The lung is very hyperæmic, and we have all the familiar signs of suffocation.

Symptoms.—In a few cases the sickness begins with certain general phenomena, such as lassitude, poor appetite and slight cough; more often, however, it appears suddenly and with severe derangement. The beginning is usually a tightness of breath amounting to asphyxia. Breathing is quickened and is often rattling, snoring and gasping. The cough is often persistent and painful; and during its attacks ribbon-shaped or tubular, sometimes even arborescent, masses of croupy matter are expectorated, up to as much as half a yard in length. Upon auscultation of the trachea and bronchi, we can hear gurgling, bubbling, quivering and rattling murmurs, and can feel tracheal fremitus with the hand. Percussion of the thorax is normal. In addition we observe signs of dyspnœa and asphyxia, viz., cyanosis of the visible mucous membranes, breathing with widely-opened mouth and out-stretched tongue, anxious look, etc. The *duration* of the attack is brief; it has usually attained its height by the second or third day and ends speedily, either with ejection of the croupy matter or with death from suffocation. Youthful animals with a small trachea usually die in this manner. The total length of the disease does not exceed six to eight days. Cattle and sheep are most liable to its attacks.

Therapeutics.—Owing to its very rapid course the treatment of bronchial croup is generally futile, the more so as even tracheotomy produces little or no beneficial result. The utmost that can be done is to try and loosen the croupy membrane by inhalation of steam or vapour of lime-water, while to smaller animals we may also give an emetic. Treatment usually comes too late, and with all severe cases in cattle early slaughtering is the wisest step. According to Trasbôt, blood-letting sometimes produces rapid improvement.

VERMINOUS BRONCHITIS. LUNG-WORM SICKNESS. HUSK. HOOSE.

Etiology.—Lung-worm sickness, or lung-worm plague, also known as lung-worm cough, is caused by animal parasites of the nematode or thread-worm order and by some species of strongylus (palisade-worms), which penetrate to the trachea and bronchi and produce at first a verminous bronchitis and catarrhal pneumonia, and later a verminous inflammation of the lungs. In our several domestic animals the following strongyli are of clinical importance:

1. In **sheep** and **goats** chiefly *strongylus filaria*. Less common is strongylus paradoxus and str. rufescens (commutatus). Besides these we must mention the hair-worm, called by Koch "pseudalius ovis pulmonalis," and by Müller "pseudalius capillaris."

2. In **cattle** *strongylus micrurus*.

3. In **pigs** *strongylus paradoxus*.

4. In **poultry** the *syngamus trachealis*.

Zoological Notes.—The lung-worms which occur in man (strongylus paradoxus) are also extremely diffused as parasites throughout the animal world. The following mammals are thus infested:

1. *Sheep:* strongylus filaria, paradoxus, commutatus, rufescens; pseudalius (strongylus capillaris). In African sheep: str. minutissimus.
2. *Goat and camel:* strongylus filaria and capillaris.
3. *Cattle:* strongylus micrurus.
4. *Pig:* strongylus paradoxus.
5. *Horse and ass:* strongylus micrurus, filaria and Arnfieldi.
6. *Dog:* strongylus canis bronchialis (Osler).
7. *Chamois and antelope:* strongylus filaria; pseudalius capillaris.
8. *Roe, stag and fallow-deer:* strongylus micrurus, filaria and sagittatus.
9. *Cat, hare, and rabbit:* strongylus commutatus and pusillus; trichosoma aërophilum; ollulanus tricuspis.
10. *Fox:* crenosoma semi-armatum; trichosoma aërophilum.
11. *Marten, polecat and weasel:* filaroides mustelarum.
12. *Dolphin:* pseudalius tumidus, convolutus, minor and reflexus.
13. *Hedgehog:* trichosoma tenue; crenosoma striatum.

We also meet with lung-nematodes in numerous *birds*, *reptiles* and *amphibia* (frog). Thus *syngamus primitivus* is found in the domestic fowl, pheasant, turkey, duck, peacock, partridge, magpie, crow, swallow, starling and woodpecker; *syngamus trachealis* in the domestic fowl, pheasant, partridge, stork, etc. Müller has compiled a detailed medical and zoological treatise on all the lung-worms at present known in the various animal species. According to his report, strongylus rufescens and str. commutatus are identical.

Natural History Notes.—In its sexually ripe state the palisade-worm inhabits the air tubes and bronchi of sheep more particularly, and after them of cattle and pigs. It is there that the eggs and embryos are produced. The thread-worms, with their eggs and embryos, are coughed up by their hosts, when the embryos complete their development in the open, probably in pools and swamps. Whether they use any intermediate host, such as insects, rain-worms or snails, for this purpose or not, is not really known, but it is highly probable. The absorption of the worm-brood into the bodies of their hosts occurs mostly through drinking-water or fodder. It is possible that the brood reaches their air-passages through the inhalation of dried mud.

Infection usually takes place in spring; after harvest, according to Gerlach, it no longer occurs. Tapken's observations prove, however, that there are exceptions to this rule; for in the marshes along the Weser and Jàde, the immigration of the brood into the cattle takes place chiefly in July and August. The strongylus micrurus requires there six to seven weeks for development, and, when the disease runs acutely, the animals—chiefly calves—may die in a further six or seven weeks. In sheep, pronounced symptoms of disease may arise in two months after the infection. According to Schulz, infection, in the case of pigs, sometimes takes place in the latter part of autumn. The outbreak of the disease follows usually in autumn, and attains its greatest spread in wet seasons, whereas in dry ones it abates and may disappear for a time. Thus on one of the Alps, Bauer saw it return after eight years, the summer being a very wet one. We see, therefore, how important water is for the development of the worm-brood. In certain damp, swampy and peaty spots lung-worm sickness is constantly present as an enzoötic malady, but it has rarely been noticed where animals are stall-fed (Kasparek). The infection of one animal by another cannot be accepted. It is probable that the brood first attain the stomach, passing thence through the paunch into the pharyngeal cavity (rumination in sheep and cattle), and through the trachea to the bronchi. There is no evidence for the assumption that they reach the lungs through the blood-stream. In the bronchi the strongyli can complete their full development. Very frequently the young strongyli advance from the bronchi into the pulmonary tissue as far as the pleura, where they often settle in large cavities (worm-nodules). Thence they wander later back

again to the bronchi, where they attain sexual maturity and full size and often accumulate in large quantities, thus producing bronchitis and bronchiectasis, as well as congestion of the larger bronchial tubes, with occasional sudden death from suffocation. From the bronchi they are then coughed out with their eggs and embryos, and the circle of their existence is thus completed.

In contrast to this emigration of the pulmonary thread-worms (strongyli) from the lungs, the pulmonary hair-worms (pseudalius capillaris) remain behind in the lung-tissues, where they become encapsuled and die.

The vitality of palisade-worms and their embryos is very great. Thus Colin saw embryos which issued from the bursting bodies of sexually ripe strongyli and remained alive in water for over two months. Ercolani reports that the parasites were not destroyed after being dried for 30 days and then subjected for eight days to the action of spirit or solution of sublimate. Railliet states that the young embryos are capable of development after having been dried for eight months. On the other hand, Perroncito found, in his researches, that strongylus micrurus died in a 1 per cent. carbol solution after 5 minutes; in a 1 per cent. creosote solution, after 7–8 minutes; in a saturated thymol and salicyl solution after from 6–8 up to 17 minutes; and in a 1 per cent. formalin solution, after 5–6 minutes.

Occurrence.—As a complaint attacking flocks, verminous bronchitis sometimes works great havoc among sheep. Carnet reports that on the frontiers of Morocco half the entire sheep and one-third of all cattle died during an epidemic. In 1883–86, according to Wernicke, several million sheep, three-fourths of all flocks, perished of this disease in Buenos-Aires. It occurs much more rarely in enzoötic form among cattle. Tapken states that the lung-worm disease breaks out almost every year among the cattle in the marshes of the Weser and Jade (Oldenburg), and causes considerable losses, calves being especially attacked. In Russia also, according to Schulz, the lung-worm disease is very rife among pigs throughout many of the provinces. Thus, for example, in 1897, 14 per cent. of the pigs slaughtered in St. Petersburg, and 21 per cent. of those slaughtered in Moscow, were affected with the strongylus paradoxus; and 48 per cent. of those in Riga in 1898. But sporadically we very often find lung-worms in domestic animals

without any general injury being caused. Thus in Berlin, for example, during the years 1833–1888, no less than 9,680 pigs and 1,675 sheep were found to be affected by lung-worm disease; but in all these cases only the lungs were condemned by the meat-inspectors. According to Hertwig's report for 1887–8, of 275,049 slaughtered sheep there were 788, and of 419,848 slaughtered swine, 3,237 affected by strongylus in the lungs. Owing to the malignancy of the disease, it is regarded in some countries as one against which legal warranty can be demanded, the term in Austria being two months.

Anatomical Conditions.—The changes effected by strongyli in the bronchi and lung-tissues are of a varied nature. Sometimes they are the phenomena of chronic bronchitis with bronchiectasis, at others we observe lobular catarrhal-pneumonic foci, or perhaps new growths like nodules, the so-called pseudo-tuberculous pneumonia or nematode-tuberculosis. In goats, according to Bugnion, even diffuse pneumonia occurs, caused by the eggs and embryos of palisade-worms.

1. *Verminous bronchitis* consists in much swelling, as well as suppurative and hæmorrhagic inflammation of the bronchial mucous membrane, in which the embryos are sometimes found enveloped in clusters. The trachea and bronchi are puffed out like bags where the worms have lodged, and where they are found rolled up into balls and surrounded by mucous and pus. The lumen of the bronchi is full of a tough, purulent, and sometimes bloody mucus, in which both eggs and very active embryos are found. The lung-tissue round about the bronchiectasis is thickened, atelectatic, splenetic and partly also emphysematous; the lungs themselves being œdematous and devoid of blood.

2. The *lobular pneumonic foci* may exist with the verminous bronchitis just described. They develop as a result of the inflammation spreading to the lung-tissue in the form of circumscribed hepatised areas, which during further progress of the disease may change into atelectasis, splenisation or formation of abscesses. These changes are found especially in calves and pigs, and start from clusters of mature strongyli. In pigs these thread-worms often occur only in the apexes and on the edges of the lungs and, according to Kohlhepp, we may suspect their presence from superficial examination, whenever the apexes and edges of the lung are somewhat

thicker, or if some of its lobes do not fall together sufficiently. Sometimes we also find slight, local pleuritic changes.

3. The *pseudo-tubercular pneumonic foci* represent tubercle-like nodules of different sizes, with which the lung-tissue is often completely permeated, and which may be felt through the tissue near the sharp edge of the lungs. They sometimes produce a lumpy surface on the lungs and even pleuritic adhesions. These nodules are simply nests of worms with a connective-tissue capsule, from which, on cutting them, plugs can be pressed consisting of pus and strongylus-embryos. They are small peri-bronchitic foci of inflammation proceeding from the bronchi (bronchitis and peribronchitis chronica nodosa).

In addition to the various changes in the lungs we find, in advanced cases, symptoms of hydræmia, effusion of water into the body cavities and connective tissue, as well as general anæmia. In pigs we observe more especially gelatinous swelling of the mesenteric, peri-laryngeal and retro-pharyngeal connective tissue.

Lung Hair-worm Sickness.—Koch contrasts the lung thread-worm sickness just described with that caused by the hair-worm which he calls pseudalius ovis pulmonalis. This parasite, which also belongs to the nematodes, was seen by Brown, Cobbold and Axe as long ago as 1851, and was named "nematoideum ovis pulmonale" by Gray Sandic and Padley. Communications on this subject have also been published by Utz, Lydtin, van Tright, etc. The investigations of Müller have shown that the hair-worm also occurs in the chamois. He therefore proposes for it the more general name of pseudalius capillaris. Schlegel has demonstrated the worm in an epidemic among goats, and established the fact that it is no pseudalius, but a strongylus; he distinguishes it as strongylus capillaris.

The history of the hair-worm's development is at present not fully known, but it is probably like that of the thread-worm. Infection in any case takes place through food and drink. The worms pass from the stomach (rumination) through the pharyngeal cavity to the trachea and bronchi, whence they invariably penetrate to the lung-tissue and there become encapsuled. Only the larvæ, and not the original worms, escape from the bronchi to the open air (Müller). According to Schlegel, the embryos first pass out through the alimentary canal; a direct transmission of embryos and eggs from one animal to another does not occur. These hair-worms are fairly plentiful. According to Ranke, almost all the sheep brought to London markets are infested by the worm. Koch found it very general in a herd of sheep. Ruser found it in 19½ per cent. of all slaughtered sheep, and Motz in 4.2 per cent., and that throughout the year, the pseudalius being in the lungs. Schlegel found in the lungs of both sheep and goats, that the hair-worm and strongylus commutatus were present at the same time; sometimes also strongylus filaria. The pathological changes in the lungs are very varied, and even with the

naked eye two distinct forms of nodule, or pseudo-tuberculous pneumonic focus, can be distinguished.

1. Small, whitish-yellow nodules, the size of miliary tubercles, which on section are found to be solid and partly calcified and containing very small worms visible by the unassisted eye. Under the microscope they reveal an accumulation of white blood-corpuscles and in the centre a number of hair-like round worms rolled up into a ball. On the periphery of these smaller foci of inflammation is a capsule of connective tissue.

2. Larger, sharply-defined knobs, of yellow to yellowish-grey colour and from a lentil to a walnut in size, some of which are elastically soft, others firm and hard. Their section shows small hair-like worms which are partly brown (males) and partly white (females) amid a milk-white, frothy fluid. The larger nodules contain only caseous matter. Similar pathological changes are, according to Schlegel, found in goats (chronic bronchitis; lobular broncho-pneumonia in the form of worm-nodules, varying in size from that of a hazel-nut to a walnut; in very pronounced cases, diffuse pneumonia, also miliary, greyish-yellow nodules in the lungs).

The symptoms in sheep, according to Koch, are cough and increased secretion of mucus, but there is no expectoration of worms. Where the number of these is small, no morbid signs are perceptible; but severer cases exhibit the symptoms of pulmonary phthisis and cachexia. Ströse states that the parasite occasions no disorder in older animals, only in lambs is there cough, increased mucous secretion, emaciation and, in a few cases, periodical nose-bleeding. But the lambs also mostly recover. According to Schlegel, the lung-worm disease in goats also begins with cough, after which there appear emaciation, diarrhœa (due to the irritation caused by the embryos which had been coughed up and swallowed), anæmia, feebleness, and general dropsy. In the epidemic described by Schlegel, the course of the disease was very unfavourable. The duration was one or more months and nearly all of the kids, yearlings, and pregnant she-goats died. *Diagnosis* during life is based on proof of the eggs and embryos in the sputum. With regard to *prophylaxis*, Schlegel recommends drying of the fodder, burning of the infected organs, and, in the case of a serious epidemic, the slaughter of the whole flock.

Symptoms.—1. In **sheep** the signs of lung-worm sickness are those of severe chronic bronchial-catarrh with subsequent pulmonary phthisis. Of all domestic animals sheep sicken most frequently and severely, lambs and yearlings being especially attacked. It begins with slight cough, which soon grows stronger, croaking and gasping, but at last is very weak and faint. This cough is noticed most when the sheep are in movement, so that when driving a flock it seems as though all are coughing. There is at the same time a mucous sputum, with salivation, slavering and choking, and sometimes entire lumps of worms are coughed up, when the sheep often rub their noses on the ground. Breathing becomes

quicker, is rattling and gasping, and upon auscultation of the air-tubes and bronchi the most varied rattling sounds can be heard, complicated with a thin, mucous nasal discharge. As the ailment progresses, breathing grows more and more difficult ; the animals are very faint, eating, for instance, upon their knees ; skin and mucous membranes become paler ; the quality of the wool deteriorates daily, and death follows after weeks or months from exhaustion, unless suffocation puts an earlier termination to the malady. The ratio of mortality varies with the age of the animals, their condition and feeding from 10 to 70 per cent.

2. In **cattle** the symptoms are similar, and here, too, calves and young animals from one to one and a half years of age are most frequently affected. The ailment begins with attacks of strong coughing ; appetite and general condition being at first undisturbed. The course varies according to age and degree of infection. Calves may sometimes die of suffocation during the first week of sickness, but the condition generally lasts for several weeks. In most cases, with proper dietetic treatment recovery takes place spontaneously. Sometimes a growing weakness and emaciation are developed as a result of chronic lung affection, when the cough grows fainter and breathing more rapid. In such cases the symptoms of verminous bronchitis awaken suspicions of bovine pleuro-pneumonia or tuberculosis. Cattle usually have more power of resistance, and the mortality is less with them than among sheep. During an epidemic outbreak arising from strongylus micrurus, Claes has lately observed in almost all the sick cattle, ophthalmia caused by filaria papillosa in the anterior chamber of the eye. Stall-cattle generally escape the disease.

3. Among **pigs** strongylus paradoxus does not seem to be at all rare in the lungs, but these animals appear to be much less sensitive to round worms than are cattle. Full-grown pigs very seldom suffer. The disease is somewhat common among young pigs, appearing even enzoötically. We notice that different individuals suffer in various degrees, and generally the malady abates as the animal grows. Sudden death from suffocation and œdema of the lungs occurs sometimes, though not so often as has been supposed. In very young animals chronic debility is often developed.

Strongyli in other Animals.—Here they are more rarely found. For instance, Colin discovered in the lung of a cat yellowish nodules, of a size varying from that of a millet to a hemp-seed, with strongyli ; and believed that the infection of the cats took place from the lungs of the herbivora with which those animals are, in Paris, frequently fed. Eberth also found næmatodes in the lungs of cats, with marked hyperplasia of the smooth muscular tissue around the parasites. Laulanié discovered tubercle-like nodules ("pseudo-tuberculosis") in the lungs of a *dog* caused by irritation from the eggs of strongylus vasorum, which had matured in the right ventricle and pulmonal artery, whence the embryos emigrated to the bronchioli. In a cachetic dog which died of ascites Railliet and Cadiot found severe strongylosis of the heart and lungs (numerous embryos of str. vasorum in the bronchial secretion and the trachea). Osler, Blumberg and Rabe also found a round worm like a filaria in the air-tubes and bronchi of dogs, which produced warty nodules upon the mucous membrane of the trachea and bronchi (tracheïtis verrucosa), and were from a millet-seed to a coffee-bean in size ; and, in the case described by Osler, had produced in the affected dog a distemper-like sickness. According to Drouin, filaria occur in the so-called summer distemper (plaie d'été), which are identical with the cutaneous parasites, and produce nodules in the lungs, varying in size from a millet-seed to a hazel-nut, which are often confounded with those of glanders. Finally, verminous epizoötic diseases have been observed in the roe, stag, chamois *and hare (extreme emaciation, pseudo-tuberculous lung disease) ; also* in the wild hog. In the case of the roe, a mixed infection has also been noticed (Zaufal), produced by distinct varieties of strongylus (filaria and commutatus).

Therapeutics.—In lung-worm sickness prophylaxis consists in the destruction of all infected lungs and in the avoidance, if possible, of swampy pastures in spring, a measure which unfortunately cannot always be adopted. For the disease itself no cure by drugs or treatment is at present known. It has long been the custom to use fumigations and inhalations of tar, carbolic acid, creolin, oil of turpentine, oil of cloves, chloroform, ether, formaldehyde, tobacco, etc., the animals being shut up for some time in a closed space filled with the vapour in question. These vapours are supposed to make the animals cough up the bronchial mucus and the worms it contains. Steps must be promptly taken to secure the destruction of expectorated worms. For pigs an emetic is recommended (veratrine $\frac{1}{3}$ grain). More important is good, substantial nourishment and the administration of tonics, especially preparations of iron, for the retention of strength. Of late years several practitioners (Levi, Eloire, Chetchowsky, Claes, Neimann, Schafnicky, Geiger, Wessel, etc.), have recommended the intratracheal injection of anti-parasitic remedies, especially carbolic acid and oil of turpentine, also of creolin, lugol solution, rancid

animal oil, chloroform and oil of cloves. Eloire used a mixture of olive oil 3 oz., oil of turpentine 3 oz., carbolic acid 30 mins., rancid animal-oil 30 mins., of which for three days he injected 2½ drams daily. A mixture of oil and oil of turpentine 3 oz. of each, with 1¼ drams of creolin, may be used in the same way. Dieckerhoff, Tapken and others were unable to report undoubted benefit from these injections. Peters recommends the internal administration of potassium picronitrate in a 0.2 per cent. solution (10–12½ drams for a sheep).

Other Lung Parasites.—Among others, Oppenheim has published observations on pulmonary distomatosis. He draws attention to the frequent occurrence of distoma hepaticum in the lungs of cattle (bronchitis, bronchiectasis) and considers that the hepatic parasite travels, not by the way of the blood-current, but passes with the water and fodder into the throat, from which it reaches the trachea. He believes that he has observed a broncho-pneumonia in the ox, the phenomena of which were of this nature. He surmised, too, that in those localities in which the liver parasite more frequently occurred, the lung diseases of the cattle were caused by distomata, and confirmed his diagnosis by investigation of the source.

Pentastoma larvæ have been observed by Tempel in the lungs of a goat. Flagellata (trichomonas) were found by Wieting in the lungs of a pig with lobular pneumonia; syngamus laryngeus in anæmic cattle by Carré and Traimbault; and amœbæ were observed by Blanc in the lung of a sheep, with pneumonic nodules.

CHAPTER V.

DISEASES OF THE LUNGS.

INFLAMMATION OF THE LUNGS. PNEUMONIA.

General Remarks on the Sub-division of Lung Inflammation.—In no other organ of the body do so many varieties of inflammation occur, both as regards character, seat and extent, as well as cause and course, as in the lungs.

1. According to the *pathological nature* of the exudate we may divide lung-inflammation into croupous or fibrinous, catarrhal, hæmorrhagic, suppurative, necrotic, caseous and desquamative; and may also consider inflammatory œdema as a serous form. 2. According to its *seat and extent* we distinguish between lobar, lobular and miliary pneumonia, as the larger, smaller or smallest areas of the lungs may be attacked; also interstitial and inter-lobular pneumonia, the former being marked by emigration of white blood-corpuscles into the interstitial connective tissue, with consequent proliferation of this tissue, and the latter by special affection of the inter-lobular lymph-vessels, whereby, as in bovine pleuro-pneumonia, the parenchyma of the lungs is isolated, as it were, by thickened and fully distended lymph-spaces, whence this form also bears the name of dissecting pneumonia. Finally, we distinguish a broncho-pneumonia and a pleuro-pneumonia. 3. From the etiological standpoint we also speak of a genuine, *i.e.*, a primitive idiopathic and independent pneumonia; and furthermore of infectious, glanderous, tuberculous, actinomycotic, mycotic, verminous and traumatic inflammation of the lungs; of an aspiration, an inspiration and a swallowing (due to foreign bodies) pneumonia; of hæmatogenous, metastatic, hypostatic and pleurogenous forms. Among infectious diseases during which pneumonia is developed we must name equine and bovine pleuro-pneumonia, swine-fever, distemper of dogs, influenza, pyæmia and septicæmia. Vagus-pneumonia is an aspiration pneumonia, which is artificially

produced by section of the vagus. 4. In accordance with its *course* we may subdivide inflammation of the lungs into benign and malignant, abortive, ephemeral (lasting one day), fixed, *i.e.*, confined to the spot first attacked, wandering and erratic, *i.e.*, inconstantly wandering and shifting its seat; names which all point to an analogy between pneumonia and erysipelas, Malignant pneumonia with extreme debility, was formerly called typhoid; and bilious when accompanied by icterus caused partly by decomposition of blood and partly by swelling of the liver and duodenal catarrh.

In all the various species of animals we find various forms of inflammation of the lungs. In *horses* the chief kind is contagious pleuro-pneumonia (murrain), and also the primary croupous and the catarrhal pneumonia; in *dogs* the catarrhal pneumonia of distemper; in *cattle* the tuberculous and interlobular pneumonia of bovine pleuro-pneumonia; in *pigs* the necrotic pneumonia of swine-fever; and in *sheep* verminous pneumonia.

Naturally among such a number of forms each one cannot be separately discussed. For the purposes of clinical practice we shall therefore confine ourselves to a description of the following forms:

1. Croupous (genuine, primary) pneumonia in horses, cattle and other domestic animals.
2. Catarrhal pneumonia.
3. Pneumonia due to foreign bodies.
4. Mycotic pneumonia.
5. Interstitial pneumonia.
6. Metastatic pneumonia.

I.—CROUPOUS (GENUINE, PRIMARY) PNEUMONIA.

(A). CROUPOUS, SPORADIC PNEUMONIA OF THE HORSE.

Etiology.—During recent years the question has been frequently raised whether there be a croupous pneumonia of horses apart from murrain (infectious pleuro-pneumonia)? On this question we hold the affirmative opinion, as hitherto no sure proof of etiological connection between the two has been adduced. We have no doubt that, although the majority of pneumonia cases in horses belong to murrain, still there is an inflammation of the lungs appearing sporadically in horses, and which is independent of murrain. Many other observers have adopted our view from their practical experience. Primary

croupous pneumonia of horses is analagous to the sporadic croupous pneumonia of cattle, which also is independent of bovine pleuro-pneumonia. Of known external causes two must be specially mentioned, viz., over-exertion and cold. Inflammation of the lungs evidently develops from both of these in connection with a previous active, hyperæmia of the lungs. As other causes we may add : breathing hot air or irritant vapours (smoke, chlorine, sulphurous acid), close confinement of the horses in stalls (after operations, pungent embrocations, etc.) ; also injuries (contusions of the thorax).

Pathogenesis.—The existence of a primary croupous pneumonia has recently been experimentally established by Dürck. He found in the lungs of quite healthy domestic animals, immediately after death, various pathogenic organisms (pneumo-bacillus, diplococcus pneumoniæ, staphylococcus and streptococcus pyogenes). He also succeeded in exciting inflammation of the lungs in animals by the effect of artificial exposure to cold, a true, uncomplicated, lobar, fibrinous pneumonia. He obtained the same result, too, by the intra-tracheal insufflation of a strongly irritating powder. The injurious effect of this irritation first manifested itself in an acute intensive hyperæmia of the lung. In consequence of this injury of the pulmonary tissues, the opportunity was afforded to the micro-organisms which had already been settled there, of increasing and unfolding their property of exciting inflammation. Accordingly, cold is the primary, and the influence of the disease germs the secondary, power in the pathogenesis of primary pneumonia.

Anatomical Conditions.—Croupous pneumonia generally is characterised by inflammation of the alveoli and bronchioles with formation within the alveoli of an exudate rich in fibrin which quickly hardens, whereby the parenchyma of the lungs usually remains intact after the inflammation has passed away. Croupous pneumonia is, moreover, generally lobular, *i.e.*, spread over larger divisions of the lungs, and it is the anterior lower part which is most frequently attacked. In a majority of cases the inflammation is on one side only. The pathological aspect varies with the different stages. According to custom we distinguish the following :

1. *The stage of congestion* (*engorgement*). This lasts about one day. The lungs are in a condition of extreme congestive

hyperæmia, intensely reddened and swollen, retain the impress of the fingers, crackle but slightly when cut, and present a smooth, moistly lustrous section-surface, marked injection of the capillaries and even hæmorrhages. The lumen of the alveoli or bronchioli gradually fills with a serous fluid, rich in albumen (inflammatory œdema of the alveoli), with which red blood-corpuscles are mingled, due to the hæmorrhages, and also white ones which have penetrated thither, and likewise large quantities of desquamated lung-epithelium. The interstitial tissue is gelatinous and infiltrated with œdematous fluid, and the bronchi are charged with a fine, frothy fluid. The air is thus driven out of the alveoli and bronchioli and partly also from the bronchi.

2. *The stage of red hepatisation.* This begins with coagulation of the fibrinous exudation and lasts on an average two days. As the fibrinogenous substance of the blood-plasma and the fibrino-plastic matter of the white blood-corpuscles combine to form fibrin, microscopically fine threads or nets of fibrin are produced in the stiffening exudate. The lung is thereby made heavier, sinks in water, appears swollen and enlarged, has a consistency like liver or india-rubber, and is tender and friable; but sometimes it is flabby and so soft that it almost liquefies. The section-surface is greyish-red and sometimes dark red, owing to more intense inflammation and consequent hæmorrhage, that is, there are darker patches surrounded by greyish-red hepatised tissue (appearance resembling granite); in very rare cases the section-surface is uniformly red. When there is simultaneous hypostasis, this surface is steel-blue, of spleen-like consistence and colour (splenisation). In addition the section-surface appears on darker red places to be granular, an effect caused by the fibrinous plugs in the alveoli: these granules, however, cannot be squeezed out.

3. The *stage of yellow hepatisation* is produced by discoloration of the exudate, breaking down and fatty degeneration of the cellular and fibrous elements, their dissolution and incipient resorption, as well as by emigration of white blood-corpuscles from the capillaries of the alveoli. In this stage the granules upon the section-surface can be removed by pressure.

4. The *stage of resolution* is distinguished by the final liquefaction of all decomposed exuded matter and its resorption or expectoration, the lungs being restored to their original integrity as soon as this process is completed.

In the region of the inflammatory foci the lungs are often œdematous and emphysematous. The pleura above any superficially situated pneumonic area is dull, rough and opaque, and occasionally also covered with hæmorrhages (pleuritis sicca). Fluid exudation in general rarely occurs. The bronchial mucous membrane is inflamed and has sometimes a croupy coating. The lymphatic glands of the lungs are œdematously swollen, limp and often soft and pappy; not unfrequently they also are infiltrated with hæmorrhages. The mucous membrane of stomach and bowels is often found in a state of catarrh. Liver, kidneys and heart may show fatty-degeneration. In exceptional cases, and contrary to the usual course we find necrosis and abscess-formation, and also, in chronic cases, induration and atelectasis of the lung-tissue.

Symptoms.—The aspect of primary croupous pneumonia is very similar to that of contagious pleuro-pneumonia. It generally opens with a very severe attack of fever, with temperature from 105°–107° Fahr. The fever is, during the first six days, usually continuous with only slight remissions, upon which there commonly follows a very rapid fall. The speed of the pulse varies according to the animal's constitution, fluctuating between 60 and 80, or even 100. Very often the fever begins with a long shivering fit, and is manifested by great faintness, muscular weakness, dulness of the sensorium and very red mucous membranes; appetite is suppressed but never quite lost, and evacuation is retarded. In severe cases there is also a jaundiced discoloration of the conjunctiva.

In accordance with the height of the fever and extent of the lung-inflammation, the breathing is both quickened and impeded, counting on an average 20 to 60 respirations per minute. The type is preponderatingly costal, the nostrils being widely opened. The exhaled air is higher in temperature. The animals either do not lie down at all, or else only upon the affected side, but prefer to stand with wide-spread fore legs, timidly avoiding every movement. At the same time a superficial, short and painful cough is observable, which, however, is sometimes entirely suppressed. Also we may see a reddish-yellow nasal discharge.

Percussion of the thorax reveals during the initial stage a tympanitic tone, which is seldom quite clear, but mostly somewhat dulled. The second or hepatisation stage is marked by a dull percussion-sound with much resistance under the

hammer, but on the borders of the dull resonance we obtain tympanitic sounds. The third stage produces tympanitic tones once more. This typical course of percussion fails in abortive cases only, in which, owing to the very short duration of the ailment, percussion reveals no changes or only trifling ones.

Auscultation of the lungs discovers at the outset a rustling or crepitating sound ; in the second stage a bronchial breathing, particularly during expiration ; and in the third a moist rattle. Auscultation of the healthy lung often gives a stronger vesicular breathing sound, which is also frequently rough. When the pleura is implicated as well we hear friction sounds.

In many patients the urine is often alkaline for a long time together ; but during the later course is frequently acid. On the other hand, it is usually albuminous, especially during the middle stages of the disease. Its specific gravity is somewhat high, but its chlorides are diminished (exudation into the lungs), while those of the urea are increased.

Course and Prognosis.—The usual course of croupous pneumonia is typical : congestion, hepatisation and resolution following each other at regular intervals. The malady reaches its height on an average on its fifth or sixth day ; from that point renovation and resorption of exuded matter begin ; the dulness clears sometimes in a remarkably short time, even in 24 hours, but usually by degrees : we hear rattling and rustling sounds, the cough becomes looser, appetite improves, so that after some 8 to 14 days recovery is complete. In other and not rare cases, the disease does not run all its stages, but ceases in the stage of congestion or at the beginning of hepatisation. This abortive course causes the croupous pneumonia to last sometimes only one, two or three days (ephemeral pneumonia).

Death by asphyxia occurs from development of general œdema of the lungs and especially in severe or so-called typhoid cases with general dissolution of the blood, or when the inflammation is wide-spread and accompanied by extreme weakness of heart. The course becomes *chronic* in very old and very young animals, in such as are feeble, and after previous bronchitis and bronchiectasis. In these cases chronic induration of the lungs with new formation of connective tissue may be developed, as well as permanent dulness and dyspnœa ; or suppuration with persistent fever, sometimes also with shivering fits and increase of sputum ; or finally, a necrosis of the lung-

tissue with subsequent sepsis and foul smelling breath. In such chronic cases recovery occurs either in six to eight weeks, or a chronic pulmonary phthisis (broken wind) is set up, or the case ends in death from sepsis.

The *prognosis* of croupous pneumonia is nevertheless on the whole favourable. The percentage of mortality is small.

Differential Diagnosis.—Primary croupous pneumonia of horses is distinguishable first of all from catarrhal pneumonia by its clinical course. Whereas the former is preceded by active hyperæmia of the lungs, the latter develops from a previous bronchitis. The course of catarrhal inflammation of the lungs is also irregular and frequently ends in atelectasis and induration; the inflammation-foci are thereby lobular, so that percussion usually shows only small and localised dulness.

To distinguish croupous pneumonia from pleuritis we must attend especially to the typical course of the pneumonia and the results of physical examination of the lungs. In pleuritis the line of dulness is mainly horizontal; because the pleuritic exudation, like all liquids, floats upwards, while in pneumonia this line is irregular, rising and falling. Moreover, the percussion-tone in pleuritis is much duller, the bronchial breathing sound is generally absent, and finally, the friction sounds are characteristic of pleuritis. Then also the heart-beat is strengthened in pneumonia, but in pleuritis may be weakened by pressure of the exudate.

General croupous pneumonia is distinguished from contagious pleuro-pneumonia chiefly by its non-contagiousness, and also by the fact that it sometimes attacks horses which have already had the latter disease.

Therapeutics.—As a disease of typical and benign course croupous pneumonia demands no special treatment. The chief thing is dietetic care. Besides this, a diligent application of cold-water bandages about the thorax, renewed at least every ten minutes, has a favourable effect upon the local inflammation. Only when there is a departure from the typical course need we administer any medicines. Thus when there is excessively high temperature, particularly if dealing with animals deficient in resistant strength, we may use febrifuges, and where there is great heart-weakness, digitalis may be tried. We give the latter in daily doses of 2 to 2½ drams of the powdered leaves, made into an electuary with neutral salts. Among fever-remedies quinine and alcohol are not very effective, whereas anti-febrine

and anti-pyrine, in repeated doses of ½ to ¾ oz. or more, may be cited as most efficient. From its cheapness, anti-febrine is especially useful as a fever-remedy for horses, in two or three doses of 1 oz. In cases of delayed resorption, inhalations and Priessnitz' compresses are used, with internal administration of alkalis, viz., acetate of potash made into an electuary with powdered juniper berries. In gangrene of the lungs inhalations of tar or creolin are advisable. We may rub the animals vigorously, inject cold infusions into the rectum and also apply an inunction of 6 to 8 per cent. mustard-spirits on the lateral wall of the breast, as an anti-febrile and derivative remedy. For old and very much enfeebled animals, alcohol in doses of 3 to 6 oz. given with their drinking-water has done good service from an economical point of view; but even better, if it be available, is wine in quantities of from ¾ pint to 1¾ pints. Trasbôt recommends milk in considerable quantities (up to 3 gallons a day).

(B).—CROUPOUS, NON-CONTAGIOUS PNEUMONIA IN CATTLE.

Remarks on the Relations between this Complaint and the Contagious Pleuro-Pneumonia of Cattle.—The existence of a croupous, non-contagious sporadic and acute pneumonia in cattle independent of bovine pleuro-pneumonia has long been stoutly contested, especially by Gerlach and Haubner. The former, in his work on forensic veterinary science, published in 1872, referred every case of independent pneumonia with marbled hepatisation to bovine pleuro-pneumonia. The existence, however, of such an independent and non-contagious pneumonia admits of no doubt. Whatever the origin of the many cases reported, and although bovine pleuro-pneumonia may occasionally run sporadically and acutely, there yet remain so many exactly observed and carefully described cases, that the occurrence of a primary pneumonia in cattle with bovine pleuro-pneumonia has been abundantly proved. Besides, no trustworthy evidence of an etiological connection between sporadic pneumonia and that of bovine pleuro-pneumonia has yet been adduced.

The causes of idiopathic croupous pneumonia are practically unknown. Probably they are physical or chemical irritants which reach the lungs with the breath, and it is possible that we have to do with an infectious but not contagious disease. More exact researches on these points are needed.

Anatomical Conditions.—The changes here are exactly the same as in the corresponding disease of horses. A relative divergence is caused only by the different structure of the lungs of the ox, in which the inter-lobular lymph-spaces are wider, and are therefore secondarily affected more readily, thus perhaps permitting the inflammatory process to spread more quickly from the lungs to the pleura. As in horses, the inflammation is mainly lobular, and the four stages there described are also here characteristically present. The colour, however, of the section-surface during the stage of hepatisation is not so light, but rather dark-brown to dark-red. The inter-lobular connective tissue also generally participates secondarily in the inflammation, being either œdematously infiltrated or even, though only to a very slight degree, permeated by fibrinous infiltrate; only a few white blood-corpuscles are, however, lodged in the fibrin. On the other hand, the pleura is more severely affected than in horses; with pleuritis sicca we not unfrequently find the forms of pleuritis serosa and sero-fibrinosa.

Symptoms.—These are likewise identical with the symptoms described in horses. The malady usually makes its appearance suddenly and sporadically. High infective fever, severe general symptoms, great dyspnœa, much groaning and coughing are its most important phenomena. Percussion reveals at first normal, and later tympanitic and muffled tones, and on auscultation we perceive at first crepitation, then bronchial breathing, and at last rattling sounds. Croupous pneumonia in cattle is, however, often distinguished from that of horses by its more rapid and unfavourable course. Not unfrequently the ailment ends in the stage of hepatisation on the third or fourth day.

Differential Diagnosis.—The most important question in croupous pneumonia of cattle is its differentiation from bovine pleuro-pneumonia. The following points will help in so-doing:

1. Croupous pneumonia appears to occur *sporadically* only and often in byres where bovine pleuro-pneumonia has not prevailed for many years, and where it has never subsequently been seen.

2. The *clinical aspect* of croupous pneumonia differs from that of bovine pleuro-pneumonia by its acute and even peracute course and the typical sequence of its several stages of congestion, red and yellow hepatisation and of resolution. Especially at

the beginning of croupous pneumonia, when there is already a high degree of (infectious) fever, no dulness is present, as in bovine pleuro-pneumonia, which, as a rule, runs a chronic and irregular course.

3. The ***anatomical conditions*** are essentially different in the two diseases. In croupous pneumonia, owing to the acute course of the malady, the hepatised portions of the lungs are alike in age, colour and condition ; and are consequently perfectly uniform. The interstitial lymphatic system is either not implicated in the process, or only œdematously infiltrated, or, in extreme cases, permeated by a thin, fibrinous exudation, with isolated white blood-corpuscles. On the other side, the characteristics of bovine pleuro-pneumonia are : first, the varying age and appearance of the several foci of hepatisation, and, secondly, the preponderant participation of the interlobular lymph-spaces. This last manifests itself in extreme inflammation of the interstitial lymph-vessels (lymphangioitis), with formation of thrombi inside the vessels and great inflammatory infiltration of the interstitial connective tissue, by which the latter is broadened. In consequence of this, the pleura is almost always sympathetically affected in the form of exudative fibrinous pleuritis. (Compare the Differential Diagnosis of Pleuro-Pneumonia.)

From the pneumonia which occurs in the course of deer and cattle disease, the above inflammation of the lungs is distinguished by absence of contagion.*

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* **Marbling of the Lungs.**—Great importance has always been attached to so-called marbling hepatisation as a distinguishing sign. By Gerlach this was claimed exclusively for bovine pleuro-pneumonia, whereas, according to others, it is said to be also present in non-contagious croupous pneumonia. It here becomes a question of what we understand by marbling hepatisation. Those who consider a mixture of yellow or yellowish-grey bands (infiltrated inter-lobular cellular tissue) with red patches (hepatised lung-lobules) as marbled hepatisation, will find the same in several diseases of the lungs, particularly in pneumonia medicamentaria. But if, on the contrary, we only regard as marbled hepatisation an aspect in which the various lung-lobules present varieties of age and of degrees of hepatisation (the central lobules of the affected part usually show the older changes), and in which the same are fringed and interwoven by inflammatorily infiltrated connective tissue ; then we must agree with Gerlach and admit that this condition is found only in chronic bovine pleuro-pneumonia, and not in the croupous pneumonia of cattle running with acute course. The variously coloured, brownish, yellowish and grey patches, surrounded by interstitial bands, are therefore alone the decisive indications.

Therapeutics.—Owing to the rapid course of croupous pneumonia, medicinal treatment is seldom required. It is, however, naturally identical with that indicated for the similar condition in horses. Cold wrappings and infusions, as well as administration of anti-pyretics, especially alcohol, being equally appropriate for cattle.

(C).—CROUPOUS PNEUMONIA IN THE SMALLER DOMESTIC ANIMALS.

1. In **dogs**, according to Röll, croupous pneumonia is frequent and usually attacks the posterior lobes of the lungs. We cannot, however, confirm this statement, as we find that true croupous, lobar pneumonia is not common among dogs, and, in comparison with the pneumonia of distemper, is somewhat rare. The clinical aspect and pathological condition correspond exactly with those described in the horse. The most common form of pneumonia in dogs is the catarrhal.

2. In **pigs** the complaint is relatively commoner, especially among such as are driven long distances in great heat or during severe cold. But, as compared with infectious pleuro-pneumonia (swine-plague), the malady seems to be rare. Teetz has observed in young pigs a chronic cough resulting from inflammation of the lung, in styes, or in large herds. The inflammation of the lung arises from lying on damp and cold cement floors, and accordingly affects a large number of pigs.

3. Among **sheep** Roloff often met with acute croupous pneumonia in very young lambs, from 14 days to 3 weeks old. The inflammation almost invariably attacked the anterior lobe of one or both lungs, and the animals mostly died at the height of the disease from lung-œdema of the non-hepatised parts of the organ. Cox reports in a similar sense.

4. We have noted the disease somewhat more frequently among **cats** than among dogs.

2.—CATARRHAL PNEUMONIA. BRONCHO-PNEUMONIA.

Etiology.—The causes of catarrhal pneumonia are in general those of acute bronchitis. Besides cold, all objects which give rise to irritation and which reach the lungs with the inhaled air, such as dust, sand and other foreign objects, fungi, and infective matter must be included. Broncho-pneumonia, when due to the latter causes, often appears in an enzoötic form,

particularly among young animals (calves, lambs, sucking pigs). In contrast to this there is found the pneumonia produced by foreign bodies, which results from unwholesome draughts, especially in cattle (sporadic inflammation of the lung in cattle). Horses also develop broncho-pneumonia after prolonged close confinement, and after long voyages at sea (so-called ship-pneumonia); it has also been observed after the administration of chloroform. From acute bronchitis a broncho-pneumonia is developed either by spread of the inflammation from the bronchioli to the alveoli and inter-lobular tissue, or by aspiration of the infectious bronchial secretion into the alveoli.

The complaint appears most frequently as an accompaniment of infectious diseases, especially such as attack young and feeble animals. In the first place stands the distemper of dogs; the catarrhal pneumonia developed during its course is the most common pneumonia of dogs. It is also fairly common in cattle, and of great importance because it forms the usual foundation for the entrance and spread of the tubercle bacillus. In horses it is rarer, but not unknown. Among sheep, pigs and calves, as well as poultry, it plays an important part in the form of worm-pneumonia.

A special form of catarrhal pneumonia is the hypostatic or sinking pneumonia. It arises when, in consequence of gradually increasing weakness of heart, the flow of blood from the lungs is disturbed; its opening stage is therefore one of hyperæmia of the lungs. Hypostatic pneumonia has its seat in the lower parts of the lungs, whither the congested blood sinks by gravity. It occurs especially in weak and enfeebled individuals, and is commonest in sheep; also during chronic heart-complaints, blood-diseases, tetanus, and in horses after prolonged confinement.

Anatomical Conditions.—Catarrhal pneumonia is a strictly localised and therefore usually a lobular lung-inflammation. Sometimes there is only one isolated area, but frequently many areas get fused, and under some circumstances the broncho-pneumonia may become disseminated and present a large number of very small miliary foci of inflammation. This form of pneumonia also begins with hyperæmia and hæmorrhage, which are complicated with exudation, emigration of white blood-corpuscles and increased desquamation of epithelium. There is no excretion of fibrin. Consequently, on microscopical examination of the alveoli, they are not found filled

with a net-like coagulation of fibrin, as is the case in croupous pneumonia. According to the preponderance of the several inflammatory products, the lungs are seen on section to be at first black to dark-brown, and later, as the cellular elements increase, a greyish-red, greyish-yellow, and at last yellowish white colour. Corresponding hereto, pressure upon the alveoli and bronchioli produces at first a bloody or dull chocolate-coloured fluid, which is later greyish-red to pale grey. In contrast to the coarsely granulated section-surface in croupous pneumonia, the surface is here smoother, or at most only finely granulated. The consistence of the inflammatory foci is firm and hard, the tissue itself contains no air and projects slightly above the section-surface. The surrounding healthy lung-tissue is in a condition of compensatory hyperæmia. If the broncho-pneumonia be disseminated, we find scattered over the lungs whitish-grey pus-foci as large as a poppy or hemp-seed, or larger. Sometimes diffuse purulent infiltration of larger lung-areas is also observed.

Among the conditions which follow catarrhal pneumonia, atelectasis of the lungs is most important. It arises partly from stoppage of the bronchioli and alveoli with exuded matter. In the airless portions of the lungs hæmorrhage first sets in owing to congestion, followed by adhesion and growing together of the alveolar walls, as well as fresh growths of connective tissue with thickening of the septa (collapse-induration). The atelectatic spots are marked by a dark bluish-red colour. A slaty-grey pigment is formed out of the hæmorrhages (slaty induration). Sometimes the surface of the lungs sinks in over the site of the atelectasis. The lung-tissue round about the broncho-pneumonic foci, as well as that between them, is often vicariously emphysematous. The bronchi also not unfrequently expand and form bronchiectases with chronic inflammatory processes upon the mucous membrane. The pleura above the broncho-pneumonic foci is sometimes rough, the bronchial glands are swollen, œdematous and even purulently infiltrated. Among other consequences we must also mention suppuration and formation of ichor, as well as caseation of the lung-tissue. If foreign bodies have penetrated into the lungs, they are found covered by a more or less thick envelope of connective tissue (nodular, fibrinous induration).

Symptoms.—The phenomena of catarrhal pneumonia are at the outset those of acute bronchitis, from which it develops.

Then come a rise of bodily temperature to medium or even high fever, acceleration of respiration and also, as a characteristic sign, especially in dogs, a short, painful cough, which is therefore often suppressed. When there are larger broncho-pneumonic foci superficially placed, percussion reveals also circumscribed, lobular, often quite localised dulness, especially along the lower edge of the lungs, upon whose periphery a tympanitic tone is often heard. Contrary to the conditions in croupous pneumonia, auscultation very seldom discovers bronchial breathing, but the breathing sounds at the dulled places are either much weaker or have altogether ceased. On the other hand, several kinds of rattling noises can be heard.

The *course* of the condition may at times be very rapid, but is usually slow and chronic, extending over weeks and months. The result is either recovery after two or three weeks in consequence of resorption and expectoration of the inflammatory products, by which the fever abates, breathing and cough become easier and the rattling sounds grow moister ; or else the disease changes to chronic conditions, among which chronic induration of the lungs (lung-asthma), with its later complication, phthisis of the lungs, is the principal. Suppuration and necrosis of the lungs develop more rarely, and are known by high fever, great emaciation and weakness, increasing stupor and cessation of the cough. Death may occur acutely as a result of the inflammatory process spreading to larger divisions of the lungs, or of secondary œdema of the lungs. The prognosis is particularly bad with young and feeble animals.

Differential Diagnosis.—Catarrhal pneumonia is distinguished from the croupous form by its slower and typical course, its development from a bronchitis, its lobular dulnesses, by the absence of a rust-coloured nasal discharge, and by its greater malignity. Moreover, it is in horses the rarer form, but in dogs the common type. It may be more difficult to distinguish from capillary bronchitis (bronchiolitis) ; we cannot exactly determine the time when the latter changes into broncho-pneumonia. The decisive indications are generally higher fever, a painful cough, localised dulnesses and the absence of vesicular breathing sounds. These most valuable signs of catarrhal pneumonia are unfortunately not always fully present. For instance, it may be impossible to prove a dulness, because the broncho-pneumonic foci lie too low down or are not large enough, so that an opinion is difficult to form.

Therapeutics.—The treatment of catarrhal inflammation of the lungs agrees in the main with that of acute and chronic bronchitis. But anti-pyretic remedies are more frequently used, as well as those which have a tonic action upon the heart, and for the latter purpose we recommend, besides alcohol, especially digitalis and caffeïn. When the course is chronic, employ loosening and expectorant remedies (alkalis, neutral salts, apomorphine, ethereal oils, resins). Inhalations are also much used, especially of creolin, and likewise hydropathic fomentations. Attention must also be paid to diet.

Enzoötic Catarrhal Pneumonia in Young Pigs.—Ströse and Heine found in the slaughter-house at Hanover that nearly one per cent. of all the animals slaughtered presented broncho-pneumonic foci. The affected pigs came, in many instances, from the same styes, forty pigs belonging to one farmer being found to be so affected. Pure catarrhal pneumonia was never found in older pigs. Clinical phenomena were absent, a cough only being noticed. Yet the disease appears to favour the development of tuberculous broncho-pneumonia, and epidemic pneumonia of pigs. Anatomically, it is distinguished by the lobular foci from croupous inflammation of the lung, which engages an entire lobe. It may also take a purulent, a caseous, or a gangrenous course. The distinction between it and the epidemic pneumonia of swine, or swine plague, is not always easy (the bacteria of epidemic pneumonia are found, too, in the lungs, and also in the blood and other organs; while in swine-plague the intestine is always affected). Bacteriological examination shows the presence of bipolar bacteria (? an erratic form of the bacillus suisepticus) in the diseased portions of the lung.

Enzoötic Catarrhal Pneumonia in Lambs.—Friedberger has described an enzoötic catarrhal pneumonia in *lambs*, which had a certain likeness to swine-plague. Its signs were a feverish pneumonia, with widespread dulness of the lungs upon one side. *Post-mortem* revealed lobular catarrhal-desquamative foci of inflammation, with extensive catarrhal pneumonia and multiple caseous necrosing foci. Furthermore, pleuritis, purulent-fibrinous pericarditis and localised necrotic hepatitis and peri-hepatitis were all in evidence. The causes of the disease remained unknown. Galtier asserts that he has seen swine-plague communicated to wethers.

3.—PNEUMONIA DUE TO FOREIGN BODIES. TRAUMATIC PNEUMONIA. GANGRENE OF THE LUNGS.

Etiology.—The pneumonia produced by foreign bodies is at first nothing else than catarrhal, lobular, localised pneumonia. As it has hitherto been regarded as a separate form of the disease, owing to the peculiarity of its causes, we have here adopted the same rule purely on practical grounds. We

distinguish two kinds of this pneumonia; viz., that which is due to foreign bodies in the narrower sense, and traumatic pneumonia.

1. **Pneumonia due to foreign bodies** in the narrower sense, arises from such outside matter as enters the lungs by way of the air-tubes and bronchi. In its early development it presents a lobular catarrhal pneumonia, but soon advances to extreme conditions of inflammation, viz., to croupous exudation and finally to gangrene of the lungs. We class here the pneumonia caused by inhaling dust, hot smoke, pungent gases, by the penetration of corn-ears, twigs, pig-bristles and other foreign objects, as well as worms and mould-fungi. To this form of pneumonia belongs also that very important class of cases known as swallowing pneumonia and pneumonia medicamentaria (drenching pneumonia.)

The pneumonia caused by wrongly swallowing is commonest in horses, but also occurs in cattle. Among horses, pharyngitis, diseases of the gullet, cerebral affections and tetanus all provide occasion for the penetration of fodder into the larynx and trachea. Among cattle its most frequent cause is the paralytic form of puerperal fever (puerperal paresis). In foot-and-mouth disease also, whenever an aphthous pharyngitis develops, a swallowing pneumonia may be set up, as well as in coughing during rumination, or after large doses of physostigmine and pilocarpine. In all animals pneumonia due to foreign bodies may arise, by the breathing of the fœtus while still in the uterus, if the amniotic fluid or meconium happens to penetrate to its lungs. The lung-inflammations of new-born animals, when not of pyæmic origin, may probably be mostly traced to this cause. Moreover, the pneumonias arising from spontaneous breaking-up of retro-pharyngeal abscesses, or from the gradual settlement of putrid bronchial secretion in the lower-lying portions of the lungs, belong to this category, as does likewise the artificially produced vagus-pneumonia (paralysis of the laryngeal nerves, entrance of food into the larynx and windpipe).

More important than these is the drenching-pneumonia or pneumonia medicamentaria, especially in pigs, horses and cattle. Its cause is to be sought in the faulty administration of drugs, particularly liquids. To administer medicine through the nose, as is customary with quacks, to pull out the tongue, to close the nose, to strap up the throat tightly, to pour in too

large a quantity at once, continuously to hold up the animal's head, are all actions likely to result in pneumonia. The same is the case with the use of mixtures which require stirring (vegetable powders, infusions of flowers, irritant, nauseous or viscid and stringy fluids). In pigs we have the animal's often quite incurable obstinacy and screaming to contend against. With horses it is particularly dangerous to continue to hold up their heads to administer medicine after they begin to cough. Finally, intra-tracheal injection, which has lately become customary, may be followed by gangrenous broncho-pneumonia, especially if too concentrated a solution be used.

The further reaction of the bronchial mucous membrane and of the lung parenchyma depends upon the nature of the offending foreign body. Whereas distilled or spring water, as well as all non-irritant drugs in the form of solutions, can be re-absorbed without causing any subsequent inflammation, the intrusion of insoluble, of irritant or even corrosive substances produces various degrees of irritation, from the simple catarrhal to the diphtheritic, necrotic and gangrenous.

2. **Traumatic pneumonia** belongs partly to the province of surgery. In this form of the disease the foreign body penetrates into the lungs from the outer surface of that organ. In cattle this mostly occurs during traumatic pericarditis, and in dogs through needles which work their way from the gullet to the lungs. In this latter affection pleuritis is more prominent. Here also we must mention the so-called contusion pneumonia arising after crushing the thorax by a blow or a fall, as well as lung-injuries inflicted by rib-fractures, external wounds, etc.

Gangrene of the Lungs.—Pneumonia due to foreign objects is one of the most frequent causes of gangrene of the lungs. This disease may, however, develop in other ways, and especially in horses during murrain (necrotising pneumonia), after severe, croupous pneumonia with impeded circulation, after hæmorrhagic stoppages, in connection with embolic processes (compare the paragraphs on metastatic pneumonia), with bronchi-extasis and stagnant decomposing bronchial secretion.

Anatomical Conditions.—Pneumonia due to foreign bodies presents pathologically a mixture of catarrhal, croupous, necrotic and gangrenous pneumonia (gangrene of the lungs). This last is a combination of necrosis and gangrene and therefore a putrescent or bacterial pneumonia. At first the in-

flammatory process is restricted to the bronchi, but gradually seizes the peri-bronchial connective tissue and the parenchyma of the lungs, which latter shows successively the phenomena of catarrhal, croupous (especially in cattle) and diphtheritic inflammation. The affected foci in the lungs are at first blackish-red, then greyish-red, and later a muddy yellow, possess increased resistance, are almost void of air, and project very slightly above the section-surface. It is rarely possible to detect the foreign body itself in the middle of the lobe. Gradually a suppurative infiltration is developed in the portion attacked, which breaks down the tissue and often appears in the form of multiple abscesses, which lie together in groups, readily coalesce, and become foci of gangrene when air enters from the bronchi. The drenching-pneumonias are usually bilateral and settle mostly in the neighbourhood of the centre of the lungs, *i.e.*, of the principal bronchi.

As a result of gangrene the lung-tissue changes into a blackish or grey, tinder-like, greasy substance, at first solid, but later of a liquid character, so that at last the lung parenchyma is replaced by a discoloured, fœtid ichor, which contains the several products of decomposition of the albumen and fat (leucine, tyrosine, fatty acids, ammonia, sulphuretted hydrogen); and also of organic constituents, enormous numbers of putrefactive bacteria, shreds of parenchyma, pus-corpuscles, detritus, crystals of triple phosphate, etc.

The lung-tissue around these gangrenous foci is in a state of hyperæmia and œdema. In horses we also occasionally find a characteristic swelling of the inter-lobular connective tissue, in the form of broad, yellow, gelatinous bands, analogous to bovine pleuro-pneumonia. The pleura often shows a purulent or hæmorrhagic-ichorous inflammation; moreover, pneumothorax and pyo-pneumo-thorax are not rare. If the gangrene lasts long, a demarcatory lung-inflammation is formed around the gangrenous focus. The mucous membrane of trachea and bronchi is sometimes purulently or ichorously inflamed, the healthy lung is often emphysematous, and we have even observed sub-pleural, mediastinal and also subcutaneous emphysema. The rest of the body shows the aspect of sepsis or pyæmia, viz., tar-like blood, swelling of the most important organs, hæmorrhage and metastases.

Symptoms.—The phenomena of pneumonia due to foreign bodies are at first those of a localised catarrhal pneumonia and

may easily be overlooked. Not until gangrene of the lungs begins to develop (lung-caverns) do the more pregnant signs appear. These are:

1. The *breath* has at first a sweetish odour, then *smells badly*, and finally has an intense stench, which is noticeable from both nostrils and is often conjoined with a discoloured, stinking nasal discharge, in which are shreds of tissue. This bad smell may in occasional cases be absent, although the gangrene be far advanced.

2. *Physical evidence of the presence of cavities.* When these exist and are not too deeply seated, percussion reveals a tympanitic note like the noise of a cracked pot or else a metallic ring. On auscultation we perceive rattling sounds, bronchial breathing, amphoric respiration and splashing.

3. *High fever, with shivering and very frequent, small pulse* (in horses from 80 to 120 a minute), combined with weakness, debility, sopor and profuse diarrhœa, all signs which are characteristic of septic fever. We have found from experience, in solitary cases only, that although extensive caverns exist in the lungs, the appetite may continue good, and there may be scarcely any fever until shortly before death.

Diagnosis.—From the foregoing the following data are necessary for the identification of lung-gangrene, viz., the proof of a gangrenous odour and of the existence of caverns. To these comes a microscopical examination of the nasal discharge for elastic lung-tissue, or for any of its parenchyma; the presence of the latter assures diagnosis in all cases. As already stated, the disease is difficult to recognise at the outset, unless its causes be known. On this point one must remember that all acute lung disorders, which develop as complications of colic, pharyngitis, inflammation of the brain, puerperal paresis, tetanus, petechial fever, diseases of the gullet, etc., are open to grave suspicion of being pneumonia due to foreign objects.

Differential Diagnosis.—It is possible to confuse pneumonia due to foreign bodies and lung-gangrene with bovine pleuro-pneumonia, bronchiectasis, phthisis, diseases of the teeth and cranial bones, and affections of the sinuses. The two last-named maladies may be known by the fact that the breath from one nostril alone smells badly. Moreover, when the teeth are diseased, it is the oral fluid which smells offensively. More difficult and often impossible is the distinction of a lung-

cavern from a bronchiectasis; but in the latter there are no elastic fibres in the nasal discharge, nor is there usually the high degree of sepsis. From epidemic pleuro-pneumonia, that due to foreign bodies can be known by its more acute course, its tendency to gangrene and the absence of true marbling.

Prognosis.—This varies with the quantity and also with the chemical and physical nature of the intruded foreign body. It is unfavourable; for by far the majority of affected animals perish. Death may occur in a few days, or sometimes not for several weeks. Cure is not rare, even when the gangrenous smell has made its appearance. It is quite possible for a gangrenous focus to be cast off by suppuration and expectorated. Such recoveries seem to occur, especially in cattle. But if once the gangrene become diffuse, instead of merely local, then recovery is hopeless.

Therapeutics.—The treatment of pneumonia caused by foreign bodies is the same as for the catarrhal form; but when gangrene has set in becomes almost fruitless. In this case we can merely try to check the gangrenous process by inhalations or by intra-tracheal injection of creolin, lysol, ichthyol, tar, creosote, carbolic acid, oil of turpentine, etc.; remedies which may also be administered internally. At the same time the septic fever must be combatted with anti-pyretics, especially camphor and alcohol. As a preventive, great care in the administration of fluid medicines is imperative, as well as the avoidance of all drenching for pigs.

4. MYCOTIC INFLAMMATION OF THE LUNGS IN MAMMALIA.

Pneumo-mycosis aspergillina.

Etiology.—Mycotic inflammation of the lungs may be produced by various fungi: mould-fungi, actinomyces, botryomyces and schizomycetes. That caused by mould-fungus is described as mycotic inflammation of the lungs in the narrower sense. Among mammalia (horses and cattle) it is mostly aspergillus fumigatus which enters their bodies with mouldy food, or by inhalation of the spores; but among birds (fowls, pigeons, geese, ducks, parrots, cage-birds, swans, pheasants and flamingoes) it is mainly aspergillus glaucus, nigrescens and fumigatus, likewise mucor racemosus and, more rarely, conoideus.

Feeble or tender individuals, or such as suffer from catarrh, appear to be most liable to infection by mould-fungus. The disease often occurs epidemically, especially among poultry. Pech once saw seven horses in one stable affected with this disease after eating mouldy, musty straw-chop. Fuller details respecting the mycotic pneumonia of poultry are given in connection with Diseases of Respiration.

Although it must be admitted that in a great many cases where mould-fungi are found in the lungs, such as in bronchiectasis, caverns and pneumonia due to foreign bodies, their presence is merely accidental and causes no pathological injury, yet it has been clearly shown in many carefully observed instances that they have an injurious effect. By mechanical and chemical irritation, as well as by consumption of the neighbouring lung-tissue, these mould-fungi cause intensely inflammatory processes in the alveoli and bronchioli and also on the mucous membrane of the bronchi, which much resemble those described in the pneumonia due to foreign bodies.

Anatomical Conditions.—In mammalia pneumo-mycosis usually presents the appearance of a nodular, suppurative pneumonia. The several nodules vary from a hemp-seed to a lentil or pea in size, and are partly spread in large numbers over the lungs and partly confluent. They are made up either of a connective-tissue capsule, with a purulent centre containing fungi, or of globular and very small foci of inflammation, the centre of which is occupied by a broad field of mould, and their periphery separated from the sound tissue by a hæmorrhagic or hepatised zone. We rarely find diffuse inflammation of the lungs, but owing to hepatisation and inflammatory infiltration of the inter-lobular connective tissue, this may in cattle have a deceptive resemblance to bovine pleuro-pneumonia (Röckl); and in horses to epidemic pneumonia (Thary and Lucet). Microscopical examination will supply the decisive data. Likewise in horses this nodular pneumo-mycosis may sometimes lead us to suspect glanders. Upon the bronchial mucous membrane we may also observe ulcers with fungoid proliferation. The pleura above the affected parts of the lung is frequently in a state of pleuritis sicca. In one case Martin demonstrated with the lung abscesses, also abscesses in the liver, which contained fungi. In a very acute course the complete aspect of pneumonia due to foreign objects may develop, with consecutive gangrene of the lungs.

Symptoms.—The phenomena correspond with those of catarrhal pneumonia. Extensive lobular dulness on percussion seems to occur more rarely. Thus in a very acute case in a horse, which was complicated with gangrene of the lungs, Pech found the dulness to extend over half the thoracic wall. The disease is usually chronic, leading to an appearance of phthisis of the lungs, with growing difficulty of breathing and emaciation. Occasionally masses of fungi can be discovered in the sputum, a fact naturally important for diagnosis.

Therapeutics.—It is generally difficult to attempt any treatment for pneumo-mycosis, seeing that its causes can rarely be known until after death. Moreover, the fungi, when once entered, cannot well be removed or destroyed. The utmost that can be done is to try inhalations of vapours of tar, carbolic acid or turpentine, although the result will probably be unsuccessful. For larger animals intra-tracheal injections of solutions of creolin, carbolic or salicylic acid or sublimate may be experimentally tried, or a sufficiently dilute Lugol's solution, as in worm or verminous pneumonia. The disease has, however, more pathological than clinical interest.

5. INTERSTITIAL PNEUMONIA. PHTHISIS OF THE LUNGS.

Etiology.—This is not an independent disease, like croupous or catarrhal pneumonia, but the new formation of connective tissue which characterises it is connected secondarily with chronic inflammatory processes in the lungs and bronchi. Among these comes first catarrhal pneumonia, which during its later course almost always shows new growth of connective tissue; then come the pneumonia caused by foreign bodies, and also chronic bronchial catarrh, with its very frequent bronchiectases. Above all there are certain infectious pneumonic affections which are accompanied by great increase of connective tissue, such as tuberculous, glanderous, actinomycotic and botryomycotic pneumonia, bovine pleuro-pneumonia and the distemper of dogs. Croupous pneumonia of horses also, leads sometimes, though very seldom, to new formation of connective tissue. In spite of its merely secondary importance, we here treat interstitial pneumonia in a separate section for the sake of greater clearness.

Anatomical Conditions.—Interstitial pneumonia consists in the formation of new connective tissue in the lungs, in

the form of nodules between the several alveoli and lobules, or round about the bronchi. The parts of the lungs thus degenerated into connective tissue are firm in texture and creak when cut with a knife; their volume is usually diminished and they are white to greyish-white in colour. The connective tissue follows a gelatinous and soft infiltration, which compresses the alveoli and bronchioli, so that their walls even grow together; it then gradually shrinks and hardens (connective-tissue induration, or sclerosis of the lungs). In consequence of this, the conducting bronchi often expand (bronchiectasis), as do also, vicariously, the unaffected alveoli (emphysema).

Symptoms.—The chief of these is dyspnœa (in horses asthma), provided that a considerable part of the lungs be lost for breathing purposes. Owing to the frequent presence of chronic bronchial catarrh, there is also often a short, superficial and faint cough. At times no physical change can be demonstrated by percussion or auscultation; because the foci are very small or scattered, or else lie in the deeper layers of the lungs. In other cases we obtain dulness and the further symptoms of catarrhal pneumonia. If of longer duration, interstitial inflammation of the lungs produces gradual emaciation and general chronic illness, combined with debility, dropsy, albuminous urine, etc.; symptoms which, with the accompanying dyspnœa, are known as lung-phthisis (phthisis pulmonum). Death in such cases occurs from exhaustion.

Treatment is usually unavailing. At best we may try alkalis or iodide of potassium.

Lung-phthisis.—This name is applied to all chronic, destructive changes in the lungs, accompanied by great loss of flesh and of strength. It is therefore a mistake to believe that only tuberculosis of the lungs can lead to lung-phthisis. In cattle (and in man) this is most frequently the case. But other causes may produce it just as easily, such as glanderous processes in the lungs, catarrhal pneumonia, pneumonia due to foreign bodies, antinomycosis, tumours in the lungs, etc.

Lung-tumours.—As such we must cite carcinoma, melanoma, sarcoma, cavernous tumours, chondro-adenoma and fibroma. They generally avoid clinical diagnosis, as they are very hard to distinguish from other thickenings of the lungs. The metastatic lung-tumours can only be diagnosed with a fair amount of probability, when certain symptoms arise in the lungs in connection with external primary tumours (carcinomata), which lead us to suspect displacement of the cells of the tumour inwards (circumscribed dulness, dyspnœa, lung-phthisis, etc.). In a case of primary lung-carcinoma in a dog we frequently observed

hæmorrhage in the lungs during life. Grammlich has noticed the same thing in horses. A. Eber found in a number of lungs, which had been removed from otherwise quite healthy slaughtered sheep, multiple firm nodules, of size varying from that of a hazel-nut to an apple, and of a greyish-white, or yellowish-white colour, which on microscopic examination proved to be adenomata. Paraciandolo has described a case of operative cure of pulmonary carcinoma by pneumectomy.

6. METASTATIC OR EMBOLIC PNEUMONIA.

Etiology.—This also is not an independent, but a secondary condition. It occurs when waste matter (emboli) is carried into the lungs from a peripheral vein containing thrombi, or from one discharging into an ichorous focus or from the heart. On becoming wedged into the capillaries such matter causes first a hæmorrhagic infarct, setting up, owing to retrograde movement, a congestive hyperæmia in the portion of lung thus cut off from the blood-stream, with accompanying hæmorrhage. Should the emboli be septically infected, this becomes a lung abscess with gangrene of the organ. The commonest causes of this condition in our domestic animals are: suppurative thrombophlebitis of the umbilical veins, of the vena saphena and other veins, and especially pyæmic processes starting from various organs, gangrenous inflammation of the feet, *e.g.*, gangrene of the fibro-fatty frog, gangrenous melanders (necrosis-bacillus), ichoro-purulent arthritis of the articulation of the pedal joint, fistulæ resulting from phlebotomy, suppurative mastitis, puerperal metritis, abscesses formed in the course of strangles in horses, ulcerous endocarditis, etc.

Anatomical Conditions.—Metastatic pneumonia is localised. The foci vary from a pea to a walnut in size, and are chiefly found on the upper surface of the lungs corresponding to the distribution of the vessels. The hæmorrhagic infarcts represent wedge-shaped (the base of the wedge turned outwards), dark-red, hard and airless foci which, if there be septic matter in their vicinity, undergo suppuration, thus finally changing the entire hæmorrhagic plug into a rounded, purulent or ichorous abscess (lung-cavern). Along with the above we find the lesions of pyæmia and septicæmia.

Symptoms.—These consist in the sudden appearance of high fever, shiverings, hard breathing and cough. Sometimes also, though not always, we can detect dulness or a hollow in the

lungs (signs of caverns). It is always important to demonstrate the primary focus. The process usually ends fatally from general pyæmia. Rarely is a cure effected after ejection of the gangrenous focus. A fibrinous pleuritis often develops in the vicinity of the necrotic focus. If the ichorous focus breaks through into the pleural cavity, a suppurative pleuritis (empyema) is then developed.

The *treatment* of metastatic pneumonia is almost useless; we may try the same methods as are suggested for gangrene of the lungs.

HYPERÆMIA AND ŒDEMA OF THE LUNGS.

Etiology.—1. **Active Hyperæmia of the Lungs** occurs especially in horses, and in those which are full-blooded and well-nourished, after over-exertion, galloping too fast, or during excessive summer heat. In other cases the cause is the inhalation of cold or over-heated air, or of pungent and irritant gases. The exclusion of larger sections of the lungs from free circulation of blood, owing to compression by exudates, may also lead to collateral active hyperæmia of the lungs. Finally, it forms the opening stage of pneumonia.

2. **Passive Hyperæmia of the Lungs** is mainly a sequel of such heart defects as produce congestion of blood in the lungs. Among these we reckon all diseases accompanied by great weakness of heart and in which a sinking or hypostatic pneumonia is developed. A similar congestion of blood in the lungs occurs when the abdomen is greatly distended by accumulation of gas in the stomach or bowels, whereby compression is exerted upon the larger vessels.

3. **Œdema of the Lungs** consists in extravasation of serum from the blood-vessels into the alveoli, bronchioli and bronchi, and evidently arises in various ways. Inflammatory œdema of the lungs represents a serous pneumonia, which develops in complication with intense active hyperæmia of the lungs during croupous pneumonia, as well as during certain infectious diseases. Many cases of acute lung-œdema in cattle and sheep seem especially to point to malignant œdema. Thus Kitt observed acute lung-œdema in a sheep in which the *post-mortem* revealed no cause for the general symptoms, but where numerous bacilli of malignant œdema were found in the juices which

escaped from the lungs. The condition of lung-œdema is caused by engorgement of blood in the lungs as well as by inflammation, especially during the death-agony, when the venous blood can no longer flow from the lungs owing to relaxation of the heart. Finally, we must assume abnormal changes and great permeability of the vascular walls as being in many cases the cause of œdema of the lungs.

Anatomical Conditions.—In *active hyperæmia* of the lungs, these organs are found very full of blood, dark-red in colour, of increased volume, consequently bloated and firmer to the touch. Blood flows copiously from the section-surface. The capillaries are fully charged and distended, so that they project far into the lumen of the alveoli. The bronchi contain much frothy mucus, which is more or less stained with blood.

In *passive hyperæmia* of the lungs, if the congestion be of long standing, we have the aspect of a so-called "engorged lung," or of brown induration. The lung is tougher, penetrated by hæmorrhages, from which later grey or black pigment-spots develop; the vessels are greatly dilated; the mucous secretions of the bronchi contain many red blood-corpuscles; and, finally, the lungs are in a state of partial splenisation.

Œdema of the Lungs is characterised by great enlargement and an enormous abundance of juice upon the section-surface, over which flows a plentiful froth of very fine bubbles, which is also found in the bronchi. In addition we find great desquamation of epithelium and extravasation of red blood-corpuscles.

Symptoms.—In the first and third of the above three forms, which occur most frequently as independent diseases, the signs usually appear suddenly and unexpectedly. The animals betray great difficulty of breathing. Respiration is quickened and impeded, the number rising to sixty, eighty and even one hundred per minute. The nostrils are widely dilated, the visible mucous membranes very red, even bleeding at the nose and frothy nasal discharge are perceived, whereby the animals grow very restless and timid and are in danger of choking. There is also a superficial, short and, at first, dry cough. In active hyperæmia percussion reveals nothing abnormal, but in œdema we have sometimes a tympanitic tone, or at least a tympanitic accessory note to the loud sound; while in congestive œdema (hypostatic pneumonia) we have dulness. In active

hyperæmia auscultation detects a much intensified vesicular breathing, and in lung-œdema the sound of crepitation, while, as death approaches, we hear bubbling and rattling noises. The pulse is very quick, full and hard, the heart-beat palpitating. If the condition terminates in death, all the breathing trouble gradually abates and is followed by a state of stupefaction. The *course* of the malady is always very rapid, not lasting usually above from twelve to twenty-four hours. Either there is prompt recovery, speedy suffocation, or a subsequent inflammation of the lungs. *Prognosis* is nevertheless not unfavourable.

Therapeutics.—Liberal blood-letting has a truly life-saving effect in active hyperæmia and in œdema of the lungs. This is one of the few diseases which call for its prompt performance. One must also attempt cutaneous derivation by friction and blisters, also give laxatives, purging clysters and cold infusions of the rectum.

PULMONARY HÆMORRHAGE. SPITTING OF BLOOD. HÆMOPTYSIS.

General Note.—Discharge of blood from the lungs is not a disease of itself, but an accompaniment and sequel of various diseases. Owing to the striking nature of its phenomena it has attracted much attention, which may perhaps justify us in devoting a special section to its consideration. The condition is not generally very common, but more so in horses and cattle.

Etiology.—In most cases of hæmoptysis the blood comes from the tissue of the lung itself, more rarely from the mucous membrane of the bronchi, seldom from the larynx. The causes are various. In horses, over-exertion plays the chief part. Dragging excessive loads, riding too far or too fast, shock and exhaustion after bolting, are all occasionally followed by a discharge of blood. Then, too, hæmorrhage of the lungs is somewhat frequently produced in horses by glanders. In cattle it is so far caused by tuberculosis as that, during the suppuration and formation of cavities in the lung-tissue, larger vessels are thereby corroded and opened. Hæmorrhage may also occur in the parenchyma of the lungs in the course of hyperæmia of that organ, particularly the passive form, and also during the contagious pleuro-pneumonia of horses. Emboli also sometimes lead to hæmorrhagic congestion and emission of blood from the

bronchioli. Hæmoptysis is caused by certain arterial diseases, rupture of aneurisms, worm-knots (strongylus armatus in horses), or ulcerative new growths in the lungs. This symptom is also observed during many infectious diseases, such as anthrax and septicæmia. Finally, traumatic pulmonary hæmorrhages appear especially after fracture of the ribs in cattle, and as a result of foreign bodies.

Symptoms.—The characteristic sign of hæmoptysis is the discharge through the nostrils and mouth, and often in large quantities, of bright-red, frothy blood, which frequently gushes out in a stream, while considerable difficulty of respiration is observed. The animal is in danger of suffocation, breathes violently and rapidly, coughs much, trembles, appears restless and troubled, sweats profusely, and when the loss is serious, begins to sway and stumble. In such cases the mucous membranes grow continually paler, the skin turns cold, the pulse is small and at last imperceptible. On auscultation we hear bubbling sounds in the trachea and bronchi. Sometimes there is only one such attack, but the bleeding is frequently renewed after several hours or days. Death may occur suddenly from loss of blood; but the animal sometimes recovers, and the bleeding ceases permanently. Respecting differentiation from the vomiting of blood compare the differential diagnosis of Hæmatemesis, Vol. I., p. 180.

Therapeutics.—Treatment is regulated entirely by the exciting cause. Blood-letting can only be adopted when this is either hyperæmia or inflammation of the lungs. In all other cases it does harm. Cold fomentation of the thorax may always be applied, and one may try the internal administration of styptic remedies: ergot, hydrastis, tannin, alum, sugar of lead, sulphate of iron, etc. Inhaling the vapour of chloride of iron and of vinegar is said sometimes to bring relief. As a prophylactic avoid every excitement or movement, too warm a stable and too hot food.

EMPHYSEMA OF THE LUNGS.

Etiology and Pathogenesis.—We have here the presence of an abnormal amount of air in the lungs, which may arise in different ways. Its causes are most frequently purely mechanical, the entire lung, especially in working horses, being

abnormally enlarged owing to excessive distension of the thorax after forced inspirations ; or it may be that only certain divisions thereof are affected; because others, being inflamed or in a state of atelectasis, are impermeable. Changes in the texture of the lung-tissue after inflammation, such as pneumonia or bronchial catarrh (inflammatory emphysema), or due to old age (senile emphysema), are also frequent causes. In many cases there appears to be a certain predisposition of the lung-tissue to destructive, parenchymatous conditions of involution, such as attenuation of the partition-walls (atrophic emphysema). That which arises from external injury (traumatic emphysema) belongs rather to the department of surgery. Finally, putrid gases (hydrogen, carburetted hydrogen, sulphuretted hydrogen), may be generated in gangrenous lung-foci, such as arise during pneumonia caused by foreign objects, infectious pleuro-pneumonia, etc., and these may emphysematously distend the lungs (septic emphysema).

The following forms of emphysema may be clinically distinguished :

1. Chronic or substantial.
2. Interstitial or inter-vesicular.
3. Acute vesicular emphysema.

1. **Chronic** or **Substantial Emphysema** arises through enlargement of the alveoli and obliteration of the alveolar septa, owing to continuous increased air-pressure combined with a degenerative change in the parenchyma of the lungs. The alveoli are first unduly distended, and then larger air-cavities are formed by the union of several such alveoli. The more the alveoli expand, the thinner their septa naturally become. According to Stömmer's measurements (to whom we owe the first exact description of a horse's lungs when in this condition), the size of these alveoli is increased tenfold, or from $\frac{1}{150}$ to $\frac{1}{16}$ inch, while their walls are correspondingly attenuated (from 8 micro-millimètres to 1 or 2 micro-mm.). The rarefied septa then represent merely small ridges projecting into the interior of the cavity. Great rarefaction is also found in the vascular system of the lungs. In consequence of increased intra-alveolar pressure the alveolar septa are exposed to compression from both sides, whereby the vessels are stretched and narrowed, while the vascular network is widened. For this reason the capillaries show a strikingly extended course during emphysema of the lungs, considerable contraction of their lumen and often

distinct atrophy. We then perceive either broken, rounded stumps, or else two pointed vessel-stumps joined together by a thin cord (vascular atrophy within the septa). This vascular atrophy is the immediate preliminary stage of septum-atrophy. The elastic fibres simultaneously disappear, and the epithelium suffers fatty degeneration. Viewed in the mass, the lung, or its affected part, is enlarged, very bulky, poor in blood and juices, pale, very soft, puffy and downy to the touch; its edges are blunt, the surface shows impressions of the ribs and retains finger-prints; when the thorax is opened the lungs do not collapse, nor does its tissue sink when cut. This obliteration of the lung-capillaries is at the same time associated with a compensatory hypertrophy of the heart.

The Relation of Emphysema of the Lungs to Asthma in Horses has been satisfactorily cleared up by the investigations of Stömmer. In the first edition of this text-book, we said that a thorough histological and anatomical investigation of the changes in the lungs in asthma of horses was necessary in view of the want of positive knowledge on the subject. Dieckerhoff had, on the ground of theoretical considerations, upheld the view that the asthmatic phenomena were in no way the principal features produced by emphysema of the lung, but by paralysis of the bronchial muscular tissue (Adams). This assertion has been effectually confuted by Stömmer. In nine asthmatical horses he invariably discovered the presence of chronic lung-emphysema and thereby confirmed the opinion previously advanced by Gerlach, Haubner, Bruckmüller and others, according to which asthma is primarily caused by emphysema of the lungs. He has shown that the lungs of horses suffering from asthma are anatomically and histologically like those of men when attacked by chronic vesicular emphysema, and that a horse's lungs are more subject to this condition than those of a man. Moreover, in horses emphysema of the lungs represents to some extent a disease resulting from their avocation (draught-horses, race-horses).

2. **Inter-vesicular or Interstitial Emphysema,** a form somewhat common in our domestic animals, arises from rupture of the alveolar walls by abnormal air-pressure and irruption of air into the inter-alveolar connective tissue and lymph-cavities. We find it most frequently upon the surface of the lungs immediately beneath the pleura (sub-pleural emphysema), which is often uplifted in blisters the size of a pea or walnut (bullous emphysema). These blisters can be easily pushed away, differing in this from substantial emphysema. The air frequently penetrates from the sub-pleural space towards the root of the lungs and between the laminæ mediastinæ, attaining finally the anterior aperture of the chest outwards as far as the looser

connective tissue about the neck, whence it can spread in the subcutaneous cell-tissue over the whole body, not even omitting the ears and tail. This inter-vesicular lung-emphysema with subsequent subcutaneous emphysema occurs very often in domestic animals, especially cattle and horses. Its causes are usually over-exertion, drawing excessive weights, especially in great heat or great cold, or in face of a high wind, exhausting races, bolting, long and wearying transport, violent coughing-fits and, in cattle, exceedingly frequent, irritant infusions and the cough thereby produced. It may also arise from medicines being swallowed the wrong way and getting into the lungs (mixtures requiring to be shaken), which may become dangerous from the perforative action of acute and acrid vegetable particles. Finally, a localised inter-vesicular emphysema occurs sometimes in difficult labours (cattle), and during the death-agony owing to increased air-pressure in the lung.

3. **Acute Vesicular Emphysema** is nothing else than simple ectasis of the alveoli, or its distension beyond the normal, without anatomical change of structure. Sometimes it spreads over the entire lung, and at others only affects certain lobes. Total vesicular emphysema occurs during the death-agony, and also as an introductory stage of the inter-lobular form. Partial emphysema often occurs, as "vicarious" emphysema, when other parts of the lungs become impermeable, especially during chronic bronchial catarrh. The several lobules are inflated, project above the rest of the lung and are very pale. This form of emphysema has no clinical importance.

Symptoms.—1. The phenomena of *chronic emphysema* of the lungs are those of asthma, viz., of chronic, and chiefly expiratory dyspnœa, which is produced on the one hand by the diminished elasticity of the lungs and accumulation of air in the distended alveoli, and on the other by atrophy of the capillary system in the lungs. During its course hypertrophy of the heart is secondarily produced. Compare the several clinical symptoms of emphysema in the next section on "Asthma."

2. The symptoms of *acute inter-lobular emphysema* of the lungs are very characteristic. The animals are suddenly stricken with severe difficulty of breathing and seem to be in danger of choking. Their respiration is anxious, groaning and laboured; cattle open their mouths wide, stretch out the tongue

and make painful movements of the muscles of the belly (expiratory dyspnœa). There is no cough, or it is short, superficial and painful. Physical examination of the lungs reveals a very loud, clear percussion ring with tympanitic after-note; auscultation shows peculiar crackling, rustling crepitations and bubbling murmurs, also dry, rattling sounds. We also very often notice the appearance of a subsequent emphysema of the skin, especially in cattle. Appetite and thirst diminish, as also does peristalsis, but there is no feverish rise of temperature. The *course* of the malady is sometimes very acute, and death from suffocation may occur in from twenty-four to thirty-six hours. In other cases it is rather chronic, and recovery takes place after weeks or months.

Treatment can only be attempted when there is also a subcutaneous emphysema. In such cases we recommend massage of the emphysematous parts of the skin, as well as compression with bandages.

Pneumatosis of Cattle.—Michels describes by this name an emphysema of the lungs appearing enzoötically among cattle in the swampy districts of the Netherlands, of the origin of which nothing certain is known. Demeester, who had previously observed it in Flanders, traces it, not without reason, to a bronchial catarrh with cough caused by cold when grazing. Various other Belgian veterinarians have also discussed the disease: as Lettinger, who has specially observed it in fattening stalls in the neighbourhood of Metz. He attributes it to chills contracted by cattle accustomed to warm stalls. On *post-mortem* examination, he found a vesicular, inter-lobular and sub-pleural emphysema.

BROKEN WIND; ASTHMA IN HORSES.

Definition.—Asthma is rather a legal than a clinical conception. It is generally defined as a chronic, non-febrile and usually incurable difficulty of breathing, which is not directly fatal, and is in no way connected with the nature of the work done. It is met with oftenest in horses, especially those above six years old, but is by no means rare in cattle, dogs and other domestic animals. Other synonyms for the disease are "hard-breathing," "broken-wind," "pursiness," "shortness of breath," etc. We shall here discuss it so far as the condition has any clinical interest.

Etiology.—1. Diseases of the Lungs.—The most important of these is chronic, substantial emphysema of the

lungs. But asthma may also develop in the course of chronic bronchial catarrh with its resultant sequels: bronchiectasis, atelectasis, connective-tissue induration of the lungs, partial formation of emphysema and peri-bronchitis. Of especial importance is capillary bronchitis (bronchiolitis) with its results. The causes of the dyspnœa are here to be sought in narrowing of the air-passages and diminution of the respiratory surface on the one hand (inspiratory dyspnœa), and on the other hand in a decrease of the elasticity of the lung owing to morbid changes therein (expiratory dyspnœa); in many cases the latter plays the chief part. Asthma is also caused by tumours in the lungs: sarcoma, carcinoma, glanderous new growth, etc.; also by adhesion of the lungs to the ribs, or by connective-tissue induration and atrophy of the lungs following on pneumonia. Finally, compression of the lungs by hydro-thorax, or by hernia of the diaphragm, and abnormal enlargement of the liver or spleen may also produce it.

2. **Diseases of the Heart.**—The most important are non-compensated valvular defects, dilatation and congenital defects in the ventricular septum. Owing to the disturbance of circulation in the lungs to which they give rise, they produce the so-called "cardiac" asthma.

Asthma.—So-called nervous or intermittent asthma is generally believed to be a spasmodic difficulty of breathing, which is chronic, but yet has periods almost free from dyspnœa, alternating with occasional spasmodic attacks. This would make this form of tight breathing identical with the chronic asthma of man. More careful investigation into this "nervous" asthma is much to be desired, especially as the conception of human asthma has not yet been fully cleared up. There are many opinions as to its causes, which shows that the term is only a collective name. The following processes seem to lie at the root of the malady: 1. Spasm of the bronchial muscles; 2. Spasm of the diaphragm; 3. Acute exudative bronchiolitis; 4. A vaso-dilatating neurosis, in which, owing to nervous causes, great hyperæmia of the bronchioli-walls with stenosis of their lumen is set up. This, probably the most important form of asthma, is reflexly caused by a pre-disposing increase of sensitiveness on the part of the respiratory nerves, and occurs in a great many pathological processes of the nasal and pharyngeal cavities, such as: polypi, proliferations of mucous membrane, hyperplasiæ of the erectile bodies of the conchæ; which in certain predisposing conditions have the effect of irritants; also in cases of idiosyncrasy, as by the inhalation of hay dust (hay-asthma, which is said to occur in horses); by certain sensations of smell (*e.g.*, by the odours of roses or violets); by great colds and heats; after the employment of certain medicaments (salicylic acid, sulphur); at certain times and in certain localities, according to

special atmospheric irritation. In a similar way, we find mention of: Asthma dyspepticum, uræmicum, diabeticum, syphiliticum, saturninum—but it is always presumed therewith that an individual predisposing irritability had already been present beforehand.

It is very probable that in horses and other domestic animals similar breathing spasms occur, but the question requires to be more fully investigated. We believe that in dogs we have seen a few cases of genuine asthma, *i.e.*, transient spasmodic dyspnœa, without any demonstrable morbid conditions in the lungs or larynx. Prümmers has noticed the same in a horse, which suffered from a painful swelling of the right upper maxilla and of the nasal mucous membrane. The dyspnœa abated as the tumour grew less. This very interesting case forms an analogy to the morbid processes of the nasal cavity which so often cause asthma in man, and which can as a rule be surgically removed.

Symptoms.—The chief phenomenon in asthma is the difficulty of breathing, which manifests itself in various ways.

1. *Greatly accelerated respiration.* In a broken-winded horse the number of respirations will increase after rapid movement for five minutes to 50–60 and more (instead of about 30), and after movement lasting 30–60 minutes to 80 or 100 (instead of 50). The breathing may also be quite superficial.

2. *Distinctly laboured breathing.* The dyspnœa is thereby much more marked in expiration than in inspiration. The former is characterised by a striking undulating or pumping action of the abdominal muscles, which contract usually in two catches, the first one short and the other longer, producing thus the so-called asthmatic furrow (double lift) behind the false ribs. At the same time there is an active movement, a rise and fall, of the flanks, with projection of the anus. Moreover, expiration lasts disproportionately longer than inspiration. Inspiratory dyspnœa manifests itself in distension of the nostrils and, in whistling asthma, also in the occurrence of inspiratory stridors and much heaving of the ribs. Expiratory dyspnœa represents a loss of elasticity in the lungs, and the inspiratory a contraction of the respiratory lung-surfaces and impediment to the entrance of air.

3. *Slow calming of respiration.* Whereas normally after half an hour of exercise the speed of respiration becomes slowed down again in from ten to twenty minutes, or after an hour's running in twenty to thirty, an asthmatical horse will require respectively half an hour, or an hour and more.

When at rest asthmatical horses often have a dull, faint cough, combined sometimes with ejection of sputum. Percussion and auscultation yield nothing positive. We have, however in

many cases of broken-winded horses detected a distinct diminution of heart-sounds (the heart being covered by the emphysematously enlarged lungs). Along with dyspnœa we may note, especially in the so-called cardiac form arising from heart defects, an intensely laborious action of the heart. Its beat is often palpitating and jerky, sometimes imperceptible; the pulse is very much accelerated (80 to 120 a minute), irregular and mostly very weak; the heart-tones which correspond with the valvular affection are not clear, but accompanied by after-sounds. Finally, the animals sweat very easily, and these outbreaks of perspiration may be extremely profuse. The general condition is frequently unfavourable, and the hairy coat looks rough and lustreless.

Course.—This is chronic and, according to season, weather, food and work done, is marked by improvement and aggravation. The degree of asthma is very varying; it changes from conditions hardly differing from the normal, and in which the serviceability of the animals is scarcely impaired, to such as render them absolutely useless for the time being.

Therapeutics.—The remedy most frequently prescribed for asthma is arsenic in small doses continued over a long time, say 1½ to 8 grains; or in the form of liquor arsenicalis (1¼ drams to 1½ oz.). An opinion as to the action of this drug is not possible, as accurate observations are lacking. The reports as to its operation vary most strikingly. Some claim that it produces much improvement, others that it is useless, and others again that it is even injurious. That arsenic after long administration can improve an animal's condition and appearance has been fully proved by experience. But that it can also successfully combat emphysema and dyspnœa has yet to be demonstrated. Probably its alleged efficacy has been based upon the improved condition of nourishment. While passing over other remedies (strychninum arsenicum, veratrin, secale cornutum, horse-chestnut, etc.), which are often commended, we would call attention to a treatment which, according to Garsault (*Parfait Maréchal*), was highly praised as long ago as 1755, viz., the withholding of drink as much as possible. This, as is well known, plays a great part in the newer treatment introduced by Oertel for chronic disturbances of circulation. The smaller the fluid contents of the body, the more easily the heart works. The withholding of beverages would therefore

be the first thing to recommend for so-called cardiac asthma, and in the second place also for all forms of tight breathing which are complicated with disturbed circulation. One might also give the alkalis for chronic bronchial catarrh, especially in the form of artificial Carlsbad salt (3 oz. three times a day, mixed with the fodder). Dietetically one must avoid bulky and dusty foods. Nutrition should be given in the form of rich and concentrated foods, and good attendance and care should be provided.

CHAPTER VI.

DISEASES OF THE PLEURA.

INFLAMMATION OF THE PLEURA. PLEURISY. PLEURITIS.

Etiology.—Inflammation of the pleura occurs primarily as an independent disease, but more frequently it appears secondarily in the course of other maladies.

Primary pleuritis is especially met with in horses and dogs. It is in many cases caused by chill, so-called rheumatic pleuritis. That cold can produce it has been established by many positive observations. The old report of Duvieusart, who saw 200 sheep stricken with pleuritis after being shorn during a very cold February, best illustrates this statement. But we must admit that in several cases of rheumatic pleuritis no previous chill can be proved. Schindelka has frequently observed it in horses after long and uninterrupted transport by rail. Sometimes it almost seems as though pleuritis broke out enzoötically under the influence of infectious or atmospheric causes. Youthful animals seem especially liable to attack. Of 600 sucking lambs, Immelmann saw 200 perish of pleuritis. Even strong and vigorous animals, such as good draught-horses, sometimes suffer from pleuritis, due, possibly, to over-exertion combined with chill.

Inflammation of the pleura may occur *secondarily* as a complication of various primary diseases. Inflammation of the lungs produces it most frequently, and particularly the infectious pneumonia occurring during the contagious pleuro-pneumonia of horses and cattle. Genuine croupous pneumonia, especially in cattle, is, however, often associated with pleuritis; the same applies also to catarrhal pneumonia, gangrene of the lungs, and to new growths, particularly cancer, in the lungs. Moreover, new growths of the mediastinum may induce chronic pleuritis. Sometimes in the course of strangles we observe

acute inflammation of the pleura in connection with a mediastinal abscess. New growths, diverticles and injuries to the gullet are very often followed by a pleuritis of ichorous character. In the same way an inflammation of the pericardium may spread to the pleura, especially if there be some foreign body present, as may also inflammation and suppuration in the bones which surround the lungs (sternum, ribs, vertebral column). Indeed, peritonitis may cause pleuritis through the lymph-canals of the diaphragm. Another secondary form is the traumatic pleuritis which arises after contusion of the thorax, penetrating wounds in the breast, fractured ribs, punctures, or foreign bodies (such as ears of corn) forcing their way from the bronchi to the pleura. Piana saw a case of pleurisy in a dog produced by the leptothrix buccalis; and Avéradère, one in a lamb which owed its origin to the cysticercus tenuicollis. Finally there may be pleuritis in pyæmia and in septicæmia; it also may occur in acute articular rheumatism and in chronic nephritis, and in foals after the use of vetches as fodder (Aronsohn). Tuberculosis and glanders may likewise lead to the development of pleuritis.

Anatomical Conditions.—From a pathological standpoint we can distinguish a series of pleuritic forms, such as the localised and the diffuse, the dry and the exudative, also adhesive pleuritis; and further, according to the character of the exudation, we have fibrinous, sero-fibrinous, serous, purulent, hæmorrhagic and ichorous pleuritis.

On the outbreak of pleuritis the sub-pleural vessels are much injected, the lymphatic-vessels of the pleura distended, the pleura itself is often infiltrated with hæmorrhages, and is then variously spotted with red, being also lustreless and opaque. A little later the pleura becomes covered with fine, yellowish white layers or shreds of fibrinous exudate, which can easily be wiped off. They consist mainly of fibrous matter, cast-off endothelial cells, white blood-corpuscles and cell-germs. If the inflammation remains at this stage, there is no further fluid exudation, and we speak of it as dry pleurisy. If no resorption of the dry exudate takes place, the exuded layers gradually change, with transformation of their white blood-corpuscles to fibroblasts and connective-tissue cells, into an inflammatory granulation-tissue containing numerous vessels, which change leads to cicatrisation and thickening, as well as to adhesion of the two pleural leaves or layers (adhesive pleuritis). In localised

pleuritis small white spots (macula lactea) and nodules are then formed, as well as tendinous bands and threads between the lungs and ribs; and in the diffuse form, larger membranous formations, with thickening of the pleura. When the inflammation lasts long, we may have indurations of exudate over an inch in thickness. These consist of swollen and degenerated tissue and may finally calcify.

Among exudative pleurites the sero-fibrinous form is commonest. In this form a considerable quantity of fluid may be secreted in a few hours. In horses we find on an average from 4 to 11 gallons in both cavities of the pleura combined. In one case as much as 155 lbs. was present. In dogs the average quantity is from a pint to a gallon, in pigs from 1¾ quarts to 2¼ gallons. The colour of this exudate varies, being yellowish-white, or yellowish-red, or yellowish-green. In the serous fluid, flocculent or flattened masses of fibrous matter is found suspended. On resorption the serum only is at first removed; the fibrinous constituents must first suffer fatty and mucous metamorphosis, or else they are drawn into sacs and remain behind, or calcify. The purely serous form is much more rare.

Purulent pleuritis (empyema) is seen most frequently after wounds, infectious conditions of the pleura and in metastases, *e.g.*, during pyæmia. The exuded matter consists here chiefly of white blood-corpuscles. Resorption only occurs in exceptional cases. The empyema rather breaks its way through the lung into a bronchus and discharges outwardly, thus frequently admitting air to the pleural cavity (pyopneumothorax); or else the pus works a path through the thoracic wall to the outside (empyema necessitatis). When the pus has been thus discharged, such conditions heal by filling up the pleural space with new growths of granulation and connective tissue.

Ichorous pleuritis is developed especially on entrance of air to the pleural sac from the bronchi or thoracic wall, also after the opening of lung-abscesses and lung-caverns in the thoracic cavity. We also frequently meet this form of pleurisy in the course of gangrenous pneumonia, particularly in the contagious pleuro-pneumonia of horses, and it is distinguished by the bad smell and discoloured quality of the exudate, as well as by the presence therein of many kinds of putrefactive bacteria.

Hæmorrhagic pleuritis is seen during hæmorrhagic diathesis or during defective resorption of the fibrinous exudate, when a large formation of new blood-vessels in the fibrin takes place,

giving rise to hæmorrhage. The exudate is here of a brown-red colour and rich in red blood-corpuscles (hæmothorax).

When there is an abundant accumulation of fluid, the lungs are pressed upwards against the vertebral column, are constricted, diminished in volume, and finally become atelectatic and of tough, hard consistence. The sound parts of the organ are correspondingly hyperæmic or emphysematous. In consequence of one half of the lungs becoming smaller, the respective half of the thorax may also grow smaller and sink in. The heart is thrust from its place, the larger vessels in the thoracic cavity are compressed, so that congestion is set up in the venous system (congested liver, kidneys, etc.), and the diaphragm is pushed back against the abdominal cavity. Pleuritis occurs more frequently on one side than on both. The communication of the two pleural sacs through the mediastinum is generally cut off early in the disease, by the fibrinous exudation.

Symptoms.—Rheumatic pleuritis usually begins with sudden shivering fits and great rise of temperature, sometimes also with other rheumatic symptoms, *e.g.*, with colic or evidences of pain in walking. The fever, which at first is somewhat high (up to 105° or 106° Fahr.), becomes later more moderate (103° to 105°), and differs from that of certain pneumonias by its irregular and intermittent character; not unfrequently we observe feverless intervals of several days. The pulse-beat is usually remarkably rapid, which arises probably from pain and also, later, from the pressure exerted by the exudate upon the heart. In horses it fluctuates between 60 and 100 beats a minute, the pulse itself being very weak and small. At the same time there is great lassitude, appetite is poor, external temperature irregularly distributed, the thoracic wall on the affected side being often perceptibly warmer; the tarsal conjunctivæ are very red. In rarer cases the disease develops gradually and slowly, so that on making a subsequent careful examination one is surprised to find an advanced stage of dulness.

With the feverish rise of temperature, the breathing also appears to be characteristically changed. In accordance with the extent of the inflammation it is quickened and impeded, the type of respiration being chiefly abdominal; for whereas the ribs remain almost motionless, the movement of the abdominal walls is greatly incr ased. Breathing, which is sometimes accompanied by plentiful exudation, amounts

occasionally to actual pumping, with a to-and-fro movement of the entire trunk. The nostrils are widely dilated, the fore feet planted far apart, or one in front of the other, the head is often rested upon some support, the animal groans in turning or when the inter-costal spaces are pressed, and either does not lie down at all, or only on the affected side. There is also usually a painful cough, but without sputum; this may, however, be absent. A physical examination of the thoracic cavity is of great importance for identification of the disease. Externally one may sometimes perceive an increased circumference on the affected side of the chest, with a flattening or even a bulging-out of the spaces between the ribs; the heart-beat is frequently displaced. In left-side pleuritis palpation reveals especially a dislocation of the heart, its beat having disappeared from the usual side and can be felt on the right side, especially in smaller animals.

At the outset of the disease *percussion* shows no changes, and, even when a fluid exudate has already gathered in the pleural cavity, its presence in the lower third of the cavity can only be proved with difficulty in horses, owing to the muscles of the chest; and, on the left side, because of the normal dulness caused by the position of the heart. On the other hand, when exudation is far advanced, the marked dulness of the percussion note in a horizontal direction is characteristic of the pleuritic exudate, as is also the circumstance that the upward horizontal limit of dulness varies with the animal's change of position, even if not in every case very quickly or completely. The increased resistance also which is noticed in percussion of the thoracic wall is likewise important for the detection of the pleuritic exudate. One sometimes obtains a tympanitic percussion-note at the beginning of pleuritis, when the elasticity of the lungs is partially lessened by a smaller quantity of exuded fluid, and later, often above the horizontal line of dulness; because the lungs, being also pressed upwards by the liquid, have lost something of their elasticity. The dulness rarely rises higher than two-thirds the height of the chest; and the entire pleural cavity is never filled by the exudate.

Auscultation reveals, both at the beginning and towards the end of pleuritis, the rubbing sounds (scraping, stroking, friction, creaking and rubbing at intervals), characteristic of the same, and which can often even be felt with the hand. With the advent of the fluid this rubbing noise disappears, and is indeed

absent in all cases of a mainly fluid-exudative character. The vesicular breathing sound also vanishes as the pleuritic exudate increases, so that one hears either an indistinct breathing, or nothing at all, or else it is finally replaced by a bronchial breathing when the lungs are only moderately compressed by the liquid; but this also is absent when such compression becomes too strong, because then the bronchi also are constricted. Sometimes one can likewise hear a bronchial breathing above the exudate, *i.e.*, above the horizontal line of dulness, in consequence of compression of the lungs, and this both during inhalation as well as expiration. The vesicular breathing noise in the sound half of the lungs is intensified; and in severe cases the ear may detect symptoms of œdema of the lungs (rustling, rattling). Along with these noises proceeding from the pleura and lungs, one may sometimes notice a dull muscular sound caused by contraction of the inter-costal muscles or of the great pectoral muscles.

Important also are the *changes in the urine*. As the exudate grows in quantity, the amount of urine becomes diminished; it loses also its chlorides. Later, as the exudate is resorbed, the chlorides reappear in the urine, while the latter becomes more plentiful, occasionally very abundant (critical discharge of urine). It moreover often contains albumen; but, according to our observations, the urine of horses often retains its alkaline reaction for a very long time: a fact we must maintain in opposition to the statements of others.

The above symptoms only occur in widely diffused pleuritis. Localised, dry pleuritis appears almost without symptoms, as is shown by the result of *post-mortem* examination.

Course and Termination.—So long as rheumatic pleuritis has not gone too far, its course is usually so favourable that complete and rapid resolution may take place. But widespread inflammation of the pleura may, on the contrary sometimes lead to death, owing to œdema of the opposite half of the lungs, or to compression of the heart. In general, pleuritis takes a dragging course, broken by sundry relapses, whereby, after a long stage of convalescence, permanent breathing difficulties appear, owing to adhesion of the lung to the pleura, as well as to the induration of the lungs which frequently remains behind. Or else the pleuritis becomes chronic, numerous feverish relapses occur, during which chronic disturbances of circulation develop in the heart beneath the pressure of the

exudate, as well as in the venous system of the body, with subsequent symptoms of dropsy, which finally pass into general emaciation and cachexia. In extreme cases the exudate at last becomes purulent, and empyema takes place; which announces itself by repeated shivering fits, a high fever of hectic type, possibly also by complication with pneumonia (pyo-pneumothorax) and rapid collapse. The *prognosis* of pleuritis is not so favourable as that of croupous pneumonia.

Diagnosis.—The diagnosis of pleuritis is assured by discovery of the rubbing sounds, the horizontal and distinct dulness, increased resistance under the percussion-hammer, abdominal breathing, diminished or dislocated heart-beat, the absence of nasal discharge and by the atypical and irregular course of the fever. In suspected cases of exudative pleuritis an exploratory puncture will remove all doubt. Although diagnosis is not difficult when the disease is far advanced, yet at its outbreak it is often hard or even impossible to differentiate, especially from pneumonia. In this case repeated and frequent examination by day is necessary, as the rubbing noises are often only heard for a short time at once. For the other signs which distinguish pleuritis from pneumonia, see the latter.

Therapeutics.—In contrast to pneumonia, with its typical course, pleuritis always demands medicinal treatment, which may be undertaken in several ways. At the outset cold packing or irrigation of the thorax is best; horses, for example, may be placed underneath a properly arranged irrigator. Strong skin-irritants upon the affected side of the chest have a similar effect, and we recommend for horses either a mustard-plaster or dilute ethereal oil of mustard (1 : 12–20 spirit, oil of turpentine, ether). By these not only is temperature reduced, but the hyperæmia is drawn towards the surface of the body, pain assuaged and resorption of the fluid exudate accelerated. Such inunctions, it is true, generally delay percussion for some days. Where the fever is very intense, administer the usual remedies: anti-febrin, anti-pyrin, etc., and especially salicylate of soda and digitalis, the two latter because they also act as diuretics. Should resorption of the exudate be delayed, one may try to expedite it either by dry fodder or by diuretic, drastic, sialagogic or diaphoretic remedies. Besides digitalis (dose for a horse ½ to 1½ drams; for a dog 1½ to 3 grains), the alkalis are also used as diuretics, especially acetate of potash, juniper berries and spirit of turpentine. Among drastics, calomel

was formerly much in use, and has been shown by later observers to have also a diuretic action. Trasbôt, for instance, gives horses an internal dose of 30 grains of calomel, and rubs in grey ointment externally. To produce salivation pilocarpin is most suitable (horses 1½ to 3 grains; cattle 3 to 6 grains: dogs up to ¾ grain), also arecolin (horses 1.25 grain), subcutaneously applied. A simple Priessnitz' compress may also assist resorption of the exudate.

Surgical treatment by means of puncture is also very important for *exudative pleuritis*. Later experience shows that it is well to effect this as soon as possible. The following are the indications for this operation, which, if performed with due aseptic precautions, is without danger: 1. When there is danger of immediate suffocation owing to the great quantity of exudate; 2. When the latter generally is very abundant; 3. When the exudate obstinately resists resorption; 4. When it is purulent or ichorous in character. In the latter case a simple puncture does not suffice, but an incision or excision of the rib must be undertaken and the pleural cavity thoroughly drained. Any injury done to the lung during puncture is of small importance. The mischievous injection of iodine into the pectoral cavity after puncture, which was formerly usual, has now been generally abandoned.

HYDROTHORAX. DROPSY OF THE CHEST.

Etiology.—This malady, which occurs in all domestic animals, and especially in dogs, is entirely secondary, and is only described here in a separate section in conformity with custom. In contradistinction to "inflammatory pectoral dropsy," as inflammation of the pleura was formerly called, it is also called "chronic" dropsy of the chest. Its causes are congestion of blood after chronic diseases of the heart, lungs and kidneys, especially valvular defects; general dropsy following some anomaly of the blood; and also increased permeability of the vessels. Along with hydrothorax we therefore often find hydropericardium and ascites. Respecting the accumulations of fluid in chronic pleuritis and in sarcomatosis and carcinomatosis of the pleura, as well as in lympho-sarcomatosis, consult the respective chapters.

Anatomical Conditions.—In contrast to pleuritis, hydrothorax is always bilateral. The fluid collected in the

pleural cavities is of clear, serous character, of yellowish, or sometimes reddish colour and without any fibrinous coagulæ. The pleura is smooth, and only somewhat loosened by maceration caused by transuded matter. The lungs are compressed in various degrees, the diaphragm pressed backwards and the intercostal spaces forwards.

Symptoms.—The chief sign of pectoral dropsy is difficulty of breathing; to this must be added physical proof of the transudate. Fever is entirely lacking. As in pleuritis, the animal's breathing is very strained, but costal respiration is not suspended, because the process is neither painful nor inflammatory. Here also percussion reveals a horizontal dulness, which, especially in small animals, at once changes place as they shift their attitude or position. Beneath the line of dulness breathing sounds can often not be heard, but sometimes bronchial respiration above it. Besides these we find also the symptoms of the primary disorder, which is most frequently a valvular defect (murmurs with the first or second sound of the heart; confluence of the two heart sounds into one), or other dropsical conditions.

Differential Diagnosis.—Hydrothorax may be known from pleuritis first of all by the absence of fever and inflammation, of the rubbing sounds, of pain in the thoracic wall and also of cough. Moreover, the flow of liquid in hydrothorax is always on both sides, while in pleuritis it is often only on one side. This fluid changes its position more easily and quickly in hydrothorax. Finally, the pleuritic exudate is mostly opaque, contains fibrin and is very rich in albumen; whereas in pectoral dropsy it is clear, without flakes of fibrin and comparatively poor in albumen. But in spite of these differences, it is sometimes hard to distinguish hydrothorax from chronic pleuritis, especially when a chronic exudative pleuritis gradually changes into dropsy of the chest. In such cases an exploratory puncture is imperative. The probability of an inflammatory process increases whenever the liquid obtained by puncture contains more than 3 per cent. of albumen and exceeds a specific gravity of 1016.

Therapeutics.—These are confined to treatment of the primary disease. Along with a dry diet, diuretics are most frequently administered, for dogs especially digitalis or diuretin. Digitalis is either prescribed alone or in combination with

acetate of potash and juniper. Diuretin is given to dogs every three hours, 4 to 15 grains in the form of powder, or in combination with digitalis, strophanthus, juniper and acetate of potash. We have also found calomel serviceable with dogs (5 doses per diem of ¾ to 1½ grains). In hydrothorax puncture should only be made when life is endangered, as the transuded matter is quickly renewed.

PNEUMOTHORAX.

Etiology.—By this term we understand the penetration of air to the pleural cavity. If there be also a pleuritic exudate, we speak of hydropneumothorax, and in case of simultaneous empyema, of pyopneumothorax. The causes of pneumothorax are various. Most commonly air penetrates from the lungs to the cavity of the chest, thus, *e.g.*, by lung-abscesses which break through into the pleural cavity and communicate with a bronchus, or when one of the lung-alveoli is broken, as is observed in cattle after interstitial empyema. In one case of an ox, Deupser was able to trace pneumothorax to the rupture of an echinococcus-cyst on the surface of the lungs, which communicated with several bronchi. Sperling has described a similar case. Fiebinger has seen pneumothorax in a tuberculous dog, as the result of the bursting of a tuberculous cavity into the pleural cavity. Stray foreign bodies, such as a grain of rye, may also finally pierce the lungs and produce pneumothorax. More rarely the air gains access from without through an injury to the wall of the thorax or from the gullet when the same has been torn. On the other hand, pneumothorax is not rarely caused in cattle by perforation of the second stomach and diaphragm by sharp foreign objects penetrating to the thoracic cavity. Finally, the generation of gas from purulent exudations in the completely closed pleural cavity may give rise to pneumothorax.

Anatomical Conditions.—True pneumothorax is rare, and occurs only in interstitial lung-emphysema of cattle. The gas here found in the pleural cavity is atmospheric air. More frequently we at the same time find serous or purulent liquid in the thoracic cavity; the air then contains but little oxygen, but much carbonic acid, carburetted hydrogen and sulphuretted hydrogen (gases of putrefaction). The lungs are compressed by this air, the heart is dislocated and the diaphragm pressed backwards.

Symptoms.—The signs of pneumo-thorax are a suddenly developed and extreme difficulty of breathing, fall of bodily temperature and symptoms of collapse. The affected half of the thorax appears enlarged, percussion reveals tympanitic and amphoric sounds, and sometimes also that of the olla rupta ; if the distension by air be very great, the tone again is loud, or has a metallic ring. On auscultation we sometimes hear as characteristic sounds a metallic splashing, amphoric respiration, as well as a noise of dropping ; but in other cases we perceive nothing, or an indistinct and occasionally bronchial breath.

Therapeutics.—The air can only be removed by puncture, but the cavity is often refilled with air from the lungs. At the same time the animal's weakness must be symptomatically combatted with stimulants (camphor, hyoscin, caffeïn, ether, alcohol).

APPENDIX TO DISEASES OF THE RESPIRATORY ORGANS.

(A). NON-PARASITIC RESPIRATORY DISEASES OF BIRDS.

Among the various kinds of poultry a simple, primary nasal catarrh is common, which arises from cold and inhalation of irritant matter, and is generally known as " Pip." It is marked by sneezing, snorting a whistling, rattling breath, opening the beak, slight dyspnœa, shaking of the head, and by a muco-purulent nasal discharge. The presence of this last can be proved by slight pressure upon the nostrils, but very often it dries up within the same to a crust. This unimportant disorder seldom demands treatment. In obstinate cases an addition of alkalis (Carlsbad salt) to the drinking-water may be prescribed. But as a precaution it is always important in cases of nasal catarrh to make sure there is no diphtheria present. In addition to rhinitis, poultry suffer from laryngitis, tracheïtis and pneumonia (croupous and catarrhal). Of especial importance is *genuine croupous pneumonia. Post-mortem* in such cases, according to Zürn, reveals great congestion of blood in the lungs, hepatisation, sub-pleural ecchymosis and extravasation, as well as repletion of the bronchi with thick, fibrous exuded matter. The symptoms consist mainly in laboured breathing with widely gaping bill, signs of pain on touching the thorax, cough and occasional expectoration of an orange-yellow mucus from beak and nostrils. In carrier-pigeons, after traversing long distances, emphysema of the skin occurs, probably in connection with lung-emphysema. But it usually passes off after a few days' rest (André).

(B). PARASITIC RESPIRATORY DISEASES OF BIRDS.

I.—POULTRY-DISEASE CAUSED BY SYNGAMUS TRACHEALIS.

Natural History.—The poultry-disease described below was first outlined by Wiesenthal in 1797 in America, where it wrought great havoc. It appeared in England in 1806, where ever since, and also in France, it has been one of the most destructive diseases of birds, especially in preserves of pheasants and poultry-yards. Year by year more than a million fowls perish of this disease in England alone. In Rothschild's pheasant-preserve in France it is reported by Mégnin that, during the prevalence of the plague, 1,200 birds were found dead every morning.

The paired wind-pipe worm (*syngamus trachealis*) belongs to the class of strongylides and occurs in the pheasant, turkey, domestic fowl, partridge, peacock, gosling and also in many other kinds of bird, such as the stork, popinjay, starling, weaver-bird, magpie, crow, swift and jackdaw. It lives in sexual maturity in pairs, the male copulatively joined to the female, in the upper part of the trachea, and mostly just beneath the rima glottidis. The female is $\frac{1}{2}$ inch long, and the male $\frac{1}{5}$ inch; their colour red, shape cylindrical, and diameter about $\frac{1}{25}$ inch. Both members of the pair attach themselves by a cup-shaped suction-disc to the mucous membrane, whence they suck blood and thus set up tracheïtis. Larger animals can bear the parasite more easily, but in smaller ones with a narrow wind-pipe, the entire lumen is often so filled up by it that they must choke. The development of the parasite is as follows: The female lays smooth, elliptical eggs, which, on being coughed out, reach the digestive canal and pass out with the dung. In warm weather eel-like embryos are developed in about eight days, the process in winter lasting some weeks. These are taken up by other birds with their food, and reach the trachea in some way at present not exactly known. There they may begin to cause cough after the seventh day.

Walker found syngamus embryos in the earth-worms of the district where the plague raged, and concluded that these form the intermediate hosts. According to Salmon, it has not been proved that earth-worms are necessary as intermediate hosts for these parasites; but he maintains that worms merely contain these embryos because they take in earth which happens to hold them. But to Walker belongs the credit of having discovered that earth-worms from a district where the disease is rife, can

communicate it to fowls which eat them. He recommends destruction of the worms in the soil of a hen-run by strong solution of salt.

Symptoms.—At first the birds appear less lively, cease taking food, often open their beaks as though yawning ("gape"). Then they begin to cough, toss their heads about, by which act whitish lumps are often thrown out. As the cough increases, breathing becomes more difficult, the creatures actually snap for air and either gradually suffocate with constantly growing tightness of breath, or else suddenly and owing to stoppage of the rima glottidis. For diagnosis it is important to prove the presence of the elliptical eggs in the fæces. Recovery only occurs in larger and older birds, or when merely solitary worms have gained entry.

Therapeutics.—Prophylaxis consists in prompt separation of sound and diseased birds, as the former may be directly infected by picking up the expectorated masses of worms; also in thorough disinfection of sheds, roosts, etc., and in burning all dead birds. Change of diet is also to be commended, with careful cleansing of all feeding vessels. Against the disease itself Mégnin advises a mixture of chopped garlic with the food, and a decoction of the same as drink. Others use inhalations of tar. Mouquet and Cordier recommend the intratracheal injection of a 5 per cent. solution of salicylate of soda as a very good remedy. Sometimes it is possible to remove the worms mechanically by means of forceps inserted through the opening of the larynx. For this purpose Cobbold performed tracheotomy.

Other Worms of the Wind-pipe.—Of other parasites which settle in the respiratory organs we must mention monostomum flavum, which occurs in the trachea of ducks; also the thread-worm of pigeons, filaria clava, found in the connective tissue between the annular cartilages of the trachea.

Syngamus bronchialis.—According to Mühlig, Przybilka, Zürn and Railliet there is found in the bronchial tubes of young geese a syngamus bronchialis which is not identical with syngamus trachealis, and is not fixed to the mucous membrane, but imbedded in glutinous, blood-red mucous masses.

2.—CYSTOLEICHUS SARCOPTOIDES—THE AIR-SAC MITE OF FOWLS.

Natural History.—This parasite inhabits especially the wind-pipe, bronchi and, above all, the so-called air-cells, particularly the abdominal cells of hens and pheasants. Viewed

by the unaided eye, the mites are round, white dots, about $\frac{1}{50}$ inch in diameter. On enlargement we see a sarcoptes-like mite, with tortoise-shaped, oval body, small oval head, and four pairs of legs, each of five members and provided with pedunculated adhesion-discs. There is possibly some genetic connection between this cystoleichus sarcoptoides and the sarcoptes cysticola, which occurs encysted in various organs of the abdominal cavity and lives under the skin. The two mites are often found together.

Anatomical Conditions.—The air-sac mite causes violent croupous inflammation in the bronchi, so that these are sometimes filled with a firm, yellowish plug. In the finer bronchi we discover the mite embedded in glairy or slimy mucus, which is often traversed by dots of blood. The lung is in places hepatised and œdematous. Occasionally the mites are found in the abdominal cells without causing any inflammation, the same being literally sprinkled white with them.

Symptoms.—The phenomena produced by the presence of air-sac mites consist in accelerated and impeded breathing. According to Zürn, the birds also emit a peculiar tone, as though there were some foreign body in the upper larynx. Otherwise they appear lively and eat well. In young chickens, Schiel observed great emaciation, in spite of a good and even ravenous appetite ; lassitude, with pronounced dyspnœa ; the duration of the disease was from 2–3 weeks. In a period of three months, all the affected birds, of which the ages ranged between six weeks and four months, died, while those of one year old and upwards did not appear to be affected. The skeleton-like emaciation was conspicuous on dissection. The walls of the air-sacs were thickly studded with white lens-shaped particles of the size of a millet-seed.

Treatment.—This is not likely to be of much effect. One must attempt to bring mite-destroying agencies to act upon the parasites through the air inhaled. To this end inhalations of the vapours of tar or tar-water are recommended. The coop must also be thoroughly disinfected.

Cytoditis nudus.—This mite has been found by Edgar in the bodies of fowls which had died suddenly. The abdominal cavity was covered with a greyish yellow dust, consisting of thousands of mites. They were also present in the heart, pericardium and aorta.

3.—PNEUMO-MYCOSIS IN POULTRY.

The invasion by fungi of the lungs of birds (hens, pigeons, geese, ducks, parrots, swans, pheasants and cage-birds) arises usually from the settlement therein of aspergillus glaucus, nigrescens and fumigatus, also of mucor racemosus. These fungi attain the bodies of their victims by inhalation of dust, or by consumption of mouldy food (millet, etc.). Besides the lungs, they are found in the bronchi, air-cells and nasal cavity. Probably owing to the generation of an inflammatory toxin (phlogosin), they produce upon the mucous membranes a diphtheritic layer in the form of sheets and scales of lamellar structure, as well as thickened, lumpy and caseous masses of pus, which contain the mycelium of the fungus and the conidiæ. Mycosis is set up in the lungs in nodular form, as in the mammalia.

The essential *symptoms* of this disease consist in accelerated, snoring respiration with rattling sounds, in diminished appetite, great lassitude (drooping the wings, closing the eyes), in wasting almost to a skeleton and finally in profuse diarrhœa, death usually following after a sickness of several weeks. Observations made in Belgium and France show that the disease may be transmitted from pigeons to man (breeders of the same), causing a malady like tuberculosis of the lungs.

Infusoria.—Willach reports upon an enzoötic among pigeons caused by infusoria. The lung-tissue is hepatised and necrotic, the liver and muscles contain yellow nodules, in which are oval, greenish bodies possessing the shape and aspect of the red blood-corpuscles of birds. These Willach regards as pathogenous infusoria, and has given them the name of balantidium viride.

CHAPTER VII.

CHRONIC CONSTITUTIONAL DISEASES.

ANÆMIA AND CHLOROSIS.

General remarks on anæmia—Anæmia of sucking-pigs and young pigs—Polyæmia (plethora).

Definition.—The term anæmia means decrease in the total quantity of the blood and consequent diminution in the number of the red and white corpuscles, and in the amount of the hæmoglobin, albuminous bodies of the plasma, salts, and water. The expression chlorosis signifies decrease only in the quantity of the hæmoglobin; the respective proportions of the blood mass, albuminous bodies, leucocytes and frequently of the red blood corpuscles being often normal. Although in mankind this condition of the blood is common, and affects only females at the time of their sexual development; we have no positive proof that it occurs in our domestic animals. The word "chlorosis" is frequently used as equivalent to "anæmia" by persons who base their definition on the principal symptom instead of on the principal cause. The practice can be accepted only on the understanding that ordinary anæmia of animals is not the same disease as human chlorosis.

Etiology.—Anæmia is often congenital, and is chiefly found in puppies, kittens, foals, calves, cattle that are specially bred for early slaughter, too finely-bred pigs, young pigs, and particularly in fancy breeds of dogs. It may appear in fat animals, and in animals which have lost a considerable quantity of blood at one time or on different occasions. We may mention that in certain districts, animals are bled on several occasions before fattening them. A permanently bad system of feeding, especially on potatoes and food poor in iron, is a prolific cause

of this disease (the so-called anæmia of inanition). We often meet with a secondary, symptomatic anæmia after many acute and chronic diseases, exhausting exertion, parturition and abnormal loss of fluid. We may here refer to the remarks made on progressive pernicious anæmia (Vol. II., p. 114). We shall discuss the anæmia of sheep in a separate section (p. 118) on account of its great importance.

Symptoms.—Anæmia is recognised by the paleness of the skin and mucous membrane; the colour of the latter appearing as if washed out to almost a milky-white. There is general debility; the patient becomes fatigued by slight exertion; the action of the heart and lungs is accelerated; the pulse small; frequently the temperature is low; the cardiac sounds are sometimes masked by *bruits de diable*; the appetite is poor; and disturbances of digestion appear, with the frequent result, in newly-born animals, of death from exhaustion after a few days. The tone of the muscles becomes lowered, and the limbs show a tendency to swell. The course of anæmia is chronic, and not unfrequently ends in recovery. Pernicious anæmia (Vol. II., p. 114) usually terminates in death. Chronic anæmia of sheep is, as a rule, complicated with hydræmia, which we shall discuss later on.

Anæmia of Sucking-pigs and Young Pigs.—Braasch states that there occurs among young pigs in Schleswig-Holstein a widely-spread anæmia, which yearly causes the death of hundreds of sucking-pigs on some farms, so as to endanger the entire breeding of pigs in that province. The animals are perfectly healthy and fat up to about 14 days after birth, from which time they assume a pale appearance and manifest signs of chlorosis. The anæmia is so great that the young pigs can be castrated without losing any blood. They also show well-marked symptoms of perverted appetite, general debility, dyspnœa, and palpitation of the heart. The animals quickly die if no remedies be applied, or they fall into a chronic state of ill-health, which is accompanied by eczema, cough, diarrhœa, swelling of the glands, etc. The disease is caused by excessive stimulation of the reproductive function of the sows, and by want of variety in their food. As the disease very rarely occurs, except in winter, it seems that the cold to which young pigs are often exposed in badly-managed piggeries (cemented floors without any litter) must have some etiological effect. Werner has observed a similar anæmia in 70 pigs belonging to one farmyard after feeding on red beetroot, raw and cooked.

Anæmia in Chickens.—According to Guitard, the rearing of fowl is impossible in many localities in France, as the chickens perish from anæmia. They sicken within a few days after escaping from the shell

with the symptoms of anæmia and lassitude, and die in 2 or 3 days. On dissection there is found merely a deficiency of the red corpuscles in the blood. Guitard regards the disease as infectious, and recommends iron with Peruvian bark.

Anatomical Conditions.—Besides the deficiency of blood in all the organs, the blood itself in anæmia is pale, resembles the juice of meat, and shows little or no tendency to coagulate. The heart and the large vessels are sometimes greatly contracted (phyoplasia); and the principal organs, such as the muscular tissue of the heart, liver, and kidneys, frequently undergo fatty degeneration, if excessive anæmia has been present for a considerable time.

Diagnosis.—The most important diagnostic appearance of anæmia is the paleness of the visible mucous membranes. We also find by physical examination, deficiency of hæmoglobin in the blood. Zschokke recommends for this purpose Gower's hæmoglobinometer (colour method). Estimates of the hæmoglobin in horses have been made by Schindelka.

Therapeutics.—Next to good, digestible, nitrogenous food, such as milk diet with horses, and a rational system of hygiene, the principal remedy for anæmia is iron. It is best to give it in a form that can be easily absorbed, and in small doses along with the food, or subcutaneously, when the digestion is very weak. The simplest preparation is ferrum pulveratum,* which is prescribed with common salt in 15½ grain doses for horses and cattle, and in 1½ grain doses for dogs. To smaller animals we give tincture of iron, such as tinctura ferri pomata,† in drops diluted with a sufficiency of water. For instance: tincture ferri pomatæ 5 parts; aqua destillata 250 parts; M.D.S. Of this we may give one teaspoonful twice a day to a dog. Besides iron, arsenic may be given as an alterative. For young animals, especially pigs, we recommend phosphate of lime in the form of soluble, ground-up bones.

Polyæmia (Plethora) is the opposite to anæmia. Formerly, plethora was frequently mentioned in text-books of pathology; but now it is rarely alluded to, and even its existence has often been denied. Müller and Cohnheim in particular have called attention to the fact that even a 50 to 80 per cent. addition to the blood mass by transfusions of homogeneous blood will not produce plethora. On the other hand, Bollinger pointed to the diversity in the percentage of blood in various

* Pure powdered iron. It nearly corresponds to *ferrum redactum*.—Tr.

† Tincture of malate of iron.—Tr.

kinds of animals. Thus, in a fat pig the percentage of blood as compared with the weight of the body amounted to only 2·2, and in a working horse to 13·5. These extraordinary diversities are arguments in favour of the possibility of plethora, which seems to occur most commonly at the commencement of fattening young, strong animals which have been well fed, but which have had no exercise for a long time; and perhaps in cases of sudden stoppage of the secretion of milk. This plethora is characterised by hyperæmia of the mucous membranes, a full and strong pulse and heart-beat, and tendency to congestion of the brain and lungs. In human beings, plethora most frequently occurs from irregularities in living. On *post-mortem* examinations in such cases we find hypertrophy and dilatation of the heart, large vessels, and capillaries of the large glands, associated with well-marked hyperæmia.

PERNICIOUS ANÆMIA.

Pernicious anæmia of animals—Pernicious anæmia of men.

Etiology.—Pernicious anæmia is a particularly malignant, primary or essential, and usually fatal form of anæmia in full-grown animals. Nothing positive is known as to its cause, although certain facts point to its infective or toxic origin. Zschokke having observed in horses several cases of it in one and the same stable, regards it as a stable epizoötic. Over-exertion, long-continued standing in the stable, and a previous attack of inflammation of the lungs, may be regarded as predisposing causes. Up to the present time, about a dozen authentic cases have been seen in horses. Imminger states that he has observed the disease as an enzoötic among cattle, and Friedberger has described a case in a mule.

Severe anæmia may become secondarily developed as a consequence of illness caused by worms, chronic suppuration, etc. Under this heading we may appropriately place the ovile disease due to distoma hepaticum (liver flukes) and the dochmiasis* of dogs and cats. The strongylus contortus in sheep, and, in a few cases, the strongylus tetracanthus in foals (after immigration into the cæcum) may also cause pernicious anæmia.

* A disease due to strongyli in the intestines. In man the parasite is the strongylus duodenalis; in dogs, the dochmius trigonocephalus, which takes up its abode generally in the small intestine and cæcum; and in cats, the dochmius Balsami felis. In all these cases, the presence of the parasite sets up anæmia and various disturbances of the alimentary canal.—TR.

Anatomical Conditions.—On *post-mortem* examination we find just as in ordinary anæmia, a general bloodless condition of the tissues and a watery state of the blood. The blood shows characteristic changes, which are absent in ordinary anæmia; namely, great paleness of the red blood corpuscles and alteration in their form (poikilocytosis). They may be increased in length, may become angular, club-shaped, or biscuit-shaped, or may assume the form of a drum-stick. Also very small (microcytes) and very large (macrocytes) blood corpuscles appear in the blood. On a hæmoglobinometric examination of the blood, the proportion of the hæmoglobin is found to have decreased very considerably. Among other important changes we may mention: fatty degeneration of the vessels, muscular tissue of the heart and skeleton, and cells of the liver and kidneys; extensive hæmorrhages in almost all the internal organs, especially under the serous membrane, in the muscles, large glands of the body, etc.; swelling of the liver and spleen; cellular infiltration of the medulla of the bones; and hæmoglobin infarcts in the kidneys. No one particular organ or tissue, with the exception of the blood, is affected to any great extent.

Symptoms.—The disease usually commences with symptoms of increasing debility so gradually, that its earliest stage is usually overlooked. In other cases, respiratory catarrh with cough and nasal discharge may be the primary symptom. It also appears that the disease may begin in an acute form with high fever. Sometimes the mucous membranes are at first yellow; but they soon become more and more pale. It is a striking fact that often, even in far advanced stages of the disease, the appetite and state of nutrition continue fairly good. On the other hand, debility, incapacity to bear fatigue, and tendency to sweat may appear at an early period. A characteristic symptom of the disease is the presence of obstinate high temperature, which frequently continues intermittent for days, and which generally defies treatment. This fever is accompanied by a very considerable increase in the frequency of the pulse. The heart sounds are sometimes complicated with secondary murmurs. The blood shows the symptoms, already described, of poikilocytosis, macrocytes and microcytes. During the further course of the disease, the animal gradually loses condition, and sometimes shows signs of dropsy; emaciation increases; and death may take place with great increase of

temperature, after the disease has lasted for months, and even for years. Ostertag saw a case of death in a horse with symptoms of paralysis (hydrorachis and hydrocephalus).

Differential Diagnosis.—An exact diagnosis can be arrived at only by an examination of the blood, which should be made immediately after the blood has left its vessels; because changes similar to those met with in the disease may be produced in the blood by the addition of different fluids (as for instance, distilled water) and by the blood being kept for some time. The absence of a primary affection of any particular organ, excessive anæmia, and presence of persistent intermittent fever, the causes of which cannot be found out, are important points for consideration. In contrast to leucæmia, in which the number of leucocytes is increased, while that of the red corpuscles remains normal, we find in pernicious anæmia no essential increase in the number of the leucocytes, no swelling of the lymph glands, and no leucæmic infarcts. The presence of hæmorrhages is common to both.

Therapeutics.—Here, as in ordinary anæmia, the principal object of our treatment should be the regeneration of the blood by preparations of iron. The results, however, are generally very unfavourable. To full-grown horses we give ferrum pulveratum in doses of ½ to 1¼ drams in combination with common salt and aromatic powders, or sulphate of iron in similar doses with small quantities of carbonate of potash and aromatic vegetable powders. We may also try arsenic: With horses we commence by giving ⅓ oz. of liquor arsenicalis, and gradually increase it up to 1⅔ oz. We may combat the fever by antipyrin, antifebrin, quinine, etc. Unfortunately, however, all these antipyretic agents, even in very large doses are only too frequently found to be useless. The affected animal should not be worked during treatment.

Pernicious Anæmia of Men.—Biermer in 1868 was the first to fully investigate this disease, which consists of an atypical formation of blood, and which by its symptoms and *post-mortem* appearances is evidently identical with the pernicious anæmia of the domestic animals. We possess no exact knowledge respecting its origin; but may hazard the conjecture that it is an infective disease. We must consider as secondary forms, but not as essential pernicious anæmia, those cases in human medicine in which the disease was due to ankylostoma [strongyli duodenalis], found by Perroncito in the workmen in the St. Gothard tunnel, miners, and brickfield labourers, or to bothriocephali. Ponfick made experimental researches

respecting the pathogenesis of pernicious anæmia and succeeded in producing it artificially in animals by repeated intravenous injections of blood-dissolving agents (glycerine, pyrogallic acid, and solution of hæmoglobin). Consequently, the essence of the disease seems to be a chronic hæmoglobinæmia in which the described changes of the red blood corpuscles simply signify a separation of the colouring matter of the blood from these corpuscles. This view is strongly supported by the proof of the presence of infarcts of hæmoglobin in the kidneys, as we have seen in the case of a horse. In the process of chronic hæmoglobinæmia we find interference with the elaboration of the blood, and deficiency of oxygen, with consequent fatty degeneration of the organs of the body and rupture of the vessels. Further research into the cause of the hæmoglobinæmia of pernicious anæmia is desirable. Possibly the disease is due to bacteria or toxins.

GENERAL DROPSY (*Hydræmia*).

General remarks—Hydræmia of sheep—Hydræmia of cattle.

General Remarks.—There are three forms of hydræmia (1) A mechanical form caused by engorgement of blood due to heart failure, and to disease of the lungs, kidneys and liver; (2) an inflammatory form; (3) and an hydræmic form caused by a serous condition of the blood, and by alteration of the vessels. The last-mentioned form occurs most frequently in sheep with accompanying symptoms of distomatosis [disease caused by flukes]. Hydræmia develops in pigs and sheep during the course of anæmia without parasitic agency, as will be discussed later on. Apart from this hyperæmia of sheep, the so-called "general œdema of cellular tissue" is another hydræmia of practical clinical importance. It occurs as an independent disease in draught-oxen in sugar factories, and in other cattle, from exclusive feeding on distillers' wash, and in horses that work in sugar factories. The causes of hydræmia are on the whole the same as those of anæmia; hereditary predisposition and bad feeding being the most important factors. The disease is divided into varieties corresponding to the respective localisation of the accumulated water, as, for instance, anasarca (water in the skin); ascites (water in the abdomen); hydrothorax (water in the chest); and hydro-pericardium (water in the covering membrane of the heart). Anasarca is found at first on the lower parts of the body, as on the legs, hypogastrium, inferior surface of the chest, and scrotum. In sheep, on the contrary, it is seen on the head,

which is kept low down while grazing. The skin is swollen and doughy to the touch, and retains the impression of the fingers; but is neither painful nor of abnormally high temperature. Animals affected with hydræmia suffer from debility, shortness of breath, and indigestion; the mucous membranes are pale and sometimes œdematously swollen; and the pulse is very weak. Besides the watery, thin condition of the blood, we find in all the cavities of the body and under the skin, accumulations of hydræmic fluid, which is clear, colourless, or yellow, free from coagula, and poorer in albumen than the blood or inflammatory lymph. It coagulates only after it has been kept a considerable time. This watery transudate contains a small number of the component parts of cells, especially of endothelial cells, and a few leucocytes.

(1.) HYDRÆMIA OF SHEEP (*chlorosis, dropsy, cachexia aquosa*).

"Rot" in Sheep.—It is a peculiar fact that the name "rot" has persisted to our day in veterinary medicine—as Gerlach has pointed out in his work on legal veterinary medicine, although without result up to the present. The term "rot" was formerly applied to any disease which was characterised by emaciation, weakness, poverty of blood, and dropsy. Tuberculosis of cattle was also called "rot," under which heading recent writers have sometimes placed distomatosis, the morbid condition set up by the presence of flukes, and diseases arising respectively from strongyli in the stomach and from tænia; and at other times, only non-parasitic ovile chronic anæmia (chlorosis) and dropsy. As we have already in Volume I. discussed the diseases just mentioned, we shall here confine our attention to the non-parasitic constitutional disease which occurs epizoötically among sheep, and which manifests itself chiefly by chronic anæmia and general dropsy.

Etiology.—The causes of dropsy or chronic anæmia (chlorosis) of sheep, which is a grave disease that attacks whole flocks, may be placed under the following two headings: (1) Insufficient and damaged food, swampy, sandy or boggy pasture land, unsanitary abodes, and bad management; and (2) unfavourable climatic conditions, such as long continued wet and cold weather, badly situated grazing land, and penning the sheep on wet, cold soil. Consequently the disease is more frequent after very bad and wet seasons and inundations, than at other times. The losses therefrom are sometimes very great.

Anatomical Conditions.—The extraordinary poverty of blood is the first thing that strikes one on viewing the dead

body. The blood resembles the juice of meat. The subcutaneous tissue of the neck, abdomen, and lower surface of the chest, and the inter-muscular connective tissue are œdematously infiltrated. The affected connective tissue seems, when it has not entirely disappeared, to be gelatinous; the muscular system is relaxed and pale; the abdominal viscera are shrunken and pale; the lungs are inflated and pale; and the heart is relaxed and flabby. A clear, light yellow transudate is found in the thoracic and abdominal cavities, pericardium, ventricles of the brain, between the cerebral membranes, and in the spinal canal.

Symptoms.—The appetite greatly varies, being sometimes good for a long period, and at other times bad from the commencement. The animal is depressed and weak; drags its legs; gives way in the loins; and becomes gradually emaciated. All the mucous membranes are very pale and final y become quite white; the conjunctiva is frequently œdematously swollen; the sclerotic has a blue tinge; and a mucous discharge from the eyes is frequently present. The skin is pale, greasy, and inflated; and the wool is lustreless, brittle, and easily falls out. The frequency of the heart and pulse is increased. Œdematous swellings develop in the intermaxillary space, on the throat, inferior surface of the chest, and hypogastrium. The abdomen increases in size and becomes fluctuating (ascites). Later on, persistent exhausting diarrhœa appears; the animal becomes so weak that it is unable to move, and continually remains recumbent. Usually the patient lingers on for several months, and sometimes for even a year.

Therapeutics.—The only remedial means which is of any use is attention to hygiene and to feeding. In the first stage of the disease a cure is always possible if we can put the animal on nutritious food which, like hay and corn, contains a large proportion of nitrogen. A great number of medicines have been recommended, the most effective of which are preparations of iron, common salt, lime and aromatic stomachics. From ancient times, gentian, sweet flag [acorus calamus], wormwood, and juniper berries have been given to sheep as stomachics for the object in question. For a flock of 100 sheep we may for instance prescribe the following mixture: *Sodii chloridum* 17 oz.; *gentianæ rad. pulv.; calami rhizomat pulv. aa* 8 oz.; *ferri sulphas, pulv.* 3 oz.; *M.D.S.*, to be made up with ground barley-malt into a mass.

(2.) HYDRÆMIA OF CATTLE (*Œdema of cellular tissue*).

Etiology.—Chronic hydræmia of cattle, which is frequently observed in abattoirs as an œdema of cellular tissue, is caused by long-continued feeding with food which contains too much water, such as distillers' wash and beetroot residue. A general hydræmia which occurs in oxen and horses, and less frequently in cows, and which sometimes assumes an enzoötic type, is of great practical interest to owners of sugar factories. This disease is chiefly caused by exclusive feeding on beetroot residue, which, since the introduction of the new method of manufacture, contains only about 5 per cent. of solid matter, with 95 per cent. of water. As the proportion of albuminoids in the solid matter is only about 1 to 10; the beetroot residue contains only about ½ per cent. of albuminoids. The consumption of such food, combined with hard work, finally brings on hydræmia, which occurs far less frequently in cows; because they are little, if at all, worked, and a large amount of water is removed out of their system along with the milk.

Anatomical Conditions.—A *post-mortem* examination of oxen which have died of general dropsy reveals, in a marked manner, the pale and flaccid condition of the entire muscular system, and the absence of *rigor mortis*. The subcutaneous and inter-muscular connective tissue is infiltrated with serous fluid; the flesh is consequently watery; the cavities of the body are more or less filled with transudate; the viscera are shrivelled up, pale and devoid of fat; and the brain is œdematous. We almost always observe signs of chronic intestinal catarrh.

Symptoms.—The disease in oxen of sugar factories becomes gradually developed. The animal shows general debility and paleness of the mucous membranes; becomes thinner and thinner in spite of the possession of an excellent appetite; passes a large amount of urine the colour of water; and the coat is rough and stands on end. Later on, increasingly severe digestive troubles set in, with alternating periods of constipation and fœtid diarrhœa. The patient is salivated and manifests on the limbs dropsical swellings which impede progression and render the gait awkward and clumsy. Œdematous swellings soon appear on the hypogastrium, dewlap, and inferior surface of the chest; and dropsical effusions form in the cavities of

the body, particularly in the abdominal cavity, so that the size of the belly is greatly increased. Finally, the animal is unable to keep on its legs ; it remains persistently recumbent, and dies from exhaustion, after the disease has lasted from 3 to 6 months.

Therapeutics.—The treatment consists essentially in the removal of the causes that produce the disease, which can be cured only, as we have already said, by giving a sufficient quantity of dry food ; supposing, of course, that the change in the system of feeding has not been undertaken too late. Diuretics and aperients have been used symptomatically to combat the dropsical swellings and effusions. Compare here the respective methods of treatment of ascites and hydrothorax.

LEUCÆMIA AND PSEUDO-LEUCÆMIA.

Nature.—Leucæmia is a changed condition of the blood in which there is a well-marked and continued increase in the number of the leucocytes. Normally, the proportion which they bear to the red blood corpuscles is about 1 to 350. In leucæmia, however, the proportion rises to 1 to 50, 1 to 20, 1 to 10, and even 1 to 2. We divide leucæmia into three varieties, according to their respective starting points. These varieties may become combined with each other.

1. *The splenic form*, which is due to hyperplasia of the spleen, is said to be distinguished from the other varieties by the presence of large, multinuclear leucocytes.

2. *The lymphatic form* which arises from hyperplasia of the lymph glands of the body.

3. *The myelogenous form* which has its origin in the hyperplastic changes of the red medulla of the bones.

Occurrence.—Leucæmia occurs more frequently in our domestic animals than is generally supposed. Up to the present it has been observed chiefly in cattle, horses, dogs, pigs, and cats. It has been also found in mice, goats and poultry. Among the 70,000 dogs which were taken to the Berlin clinic in the years 1886–1894, there were 21 cases of leucæmia. In the Prussian army, in the years 1890–1895, a total of 26 horses suffered from leucæmia.

Etiology.—The origin of leucæmia in our domestic animals is as little known as it is in man; although it is generally assumed that injuries, intermittent fever, syphilis and other weakening influences are causes of the disease in human beings. Such supposed causes suggest an infective disease which begins as an infection of the blood-forming organs, namely, the spleen, lymph glands, and red medulla of the bones. The supposition of an infective cause is in no way weakened by the fact that, up to the present, experiments in transmission have given negative results. The observations made in cattle seem to indicate that leucæmia may be developed from chronic catarrh of the abomasum and uterus. Lucet and others state that they have found specific bacteria in the blood of animals suffering from leucæmia. Löwit has recently indicated the presence of protozoa in man as the cause of leucæmia (hæmamœba leucæmiæ magna and parva intranuclearis).

Infectious Leucæmia of Fowl.—According to Moore, there occurs in birds an infectious, bacterial leucæmia, which is often erroneously taken for fowl-cholera; and Dawson has demonstrated the schizomycete therein found as *bacterium sanguinarium.*

Anatomical Conditions.—Besides the increase in the number of leucocytes, which is the essential condition in this disease, the blood shows a series of other changes. It is very pale (so-called "white blood"), and stains the hands only slightly red; coagulates very slowly; and secretes between the buffy coat and the blood clots a grey stratum which resembles pus. The spleen sometimes contains almost white, purulent blood. The clots in the heart and large vessels are soft and greasy. They resemble pus, and consist almost entirely of leucocytes. In consequence of the decrease in the number of the red blood corpuscles, the specific gravity of the blood is reduced (from 1·055 to 1·040). The blood, as has been demonstrated in human leucæmic blood, contains several abnormal substances, such as formic acid, acetic acid, lactic acid, uric acid, xanthin, hypoxanthin, leucin, etc. The white blood corpuscles show various pathological changes. Only very few of them retain their normal, granular structure; the remainder being hyaline and transparent (embryonal-typhus). We may also observe nucleated, hæmoglobin-stained leucocytes which are filled with fat granules and which are on the point of decay. Transition-forms between the above-mentioned cells may be met with.

In the splenic form, the spleen frequently becomes enlarged to double or treble its ordinary size, or even more, and becomes 5 to 10 times (even 50 times) heavier than in health: Leisering found in a horse a spleen weighing 62 lbs. 13 ozs. Hellinger reports the case of a pig the spleen of which weighed 8 lbs. 2 ozs. We may mention that the normal weight of the spleen of a horse varies from 17 ozs. to 26 ozs.; and that that of a pig is about 5 oz. The edges of the spleen are blunt; its consistence is firm; and the follicles are swollen sometimes to the size of a pea; although the microscopical structure remains normal (simple hyperplasia).

In the lymphatic variety of the disease, which is often complicated with the splenic form, the lymph glands are enlarged and may swell into tumours of considerable size. Their consistence is generally soft. Frequently the lymph glands of the head, throat, extremities, thorax and abdomen, are hyperplasitic; in other cases only a few lymph glands are affected. Peyer's patches and the solitary glands of the intestine are only slightly swollen, or are normal in size. Swelling caused by congestion sometimes appears in the region of the swollen lymph glands of the extremities, throat, head and eyes. From an extreme hyperplasia of the mesenteric glands, chyle-stasis and chyle-thrombosis may occur.

The medulla of the bones often manifests signs of diffuse hyperplasia and cellular infiltration. In the so-called pyoid form of myelogenous leucæmia, it consists almost exclusively of white blood corpuscles and is like pus in appearance. In the lymphadenoid form it resembles raspberry-jelly.

Besides the described changes, we find in the various organs, as characteristic of leucæmia, leucæmic infarcts and new growths resembling lymphomata, with more or less enlargement of the affected organs, especially in the liver (Reinländer saw one case in which the leucæmic infiltrated liver of a horse weighed 92 lbs. 10 oz.), spleen, kidneys, serous membranes, mucous membranes (abomasum and uterus in cattle), uterus, urinary bladder, ovaries, thyroid glands, subcutaneous connective tissue, lungs, retinæ, tissues round the vessels, etc. The leucæmic infarcts consist of a diffuse infiltration of the tissue with white blood corpuscles, on account of which, the bronchi and blood-vessels, for instance, are surrounded with a dirty-white covering. The new growths which resemble lymphomata appear as circumscribed tumours of the same histological condition as the lymph glands. In the course of leucæmia we may frequently, though

not always, observe hæmorrhages in various organs, especially in the kidneys.

Symptoms.—The symptoms of leucæmia are not very distinctive. The first general ones are: weakness, inability to bear fatigue, difficulty in breathing, inclination to perspire from slight exertion, attacks of vertigo, and general paleness of the mucous membranes and skin. The action of the heart is usually accelerated; the pulse is frequent, small and irregular; and auscultation of the heart sometimes reveals anæmic secondary murmurs. In some cases all the lymph glands of the surface of the body become on both sides symmetrically enlarged; in other instances, only certain glands, as for example, those in the region of the pharynx. In horses we may at times notice enlargement of the spleen in the form of a swelling of the left flank. The appetite often continues normal for a long time. In the later stages of the disease it is generally capricious, and gastric disturbances, such as diarrhœa, may become developed. Towards the end, hæmorrhages occur in various mucous membranes, as for instance those of the nose, intestine and bladder; and dropsical swellings from engorgement appear in the region of the swollen lymph glands. It is characteristic of this disease that, with all these morbid changes, no organic affection is present. Consequently, a true diagnosis can be made only by a microscopic examination of the blood. For this purpose we may obtain blood from a vein by means of a fleam or lancet, or by a simple incision into the skin. It is best to examine the blood with as little delay as possible, and to refrain from adding anything to it.

As a rule, the course of the disease is chronic, and runs on for months or even years. A few acute cases have, however, been described. Owing to the uncertain indications afforded by symptoms in this complaint, the true nature of the disease has often escaped recognition until after death, in cases in which an exact examination of the blood was omitted. Affected cattle often seem to their owners quite healthy up to the appearance of hæmorrhages in the mucous membranes. Prognosis is very unfavourable.

Differential Diagnosis.—Microscopical examination of the blood enables us to diagnose with certainty leucæmia from pernicious anæmia and other blood diseases; but not always from diseases in which an increase in the number of the white

blood corpuscles may be found, without leucæmia being present, as may be sometimes the case in a state of health, although to a lesser extent, or temporarily. Thus we have cases of leucocytosis (temporary increase of the leucocytes) in pregnancy, directly after feeding and after blood-letting, and during inflammatory diseases in general. A persistent though slight increase is also present in other diseases of the blood, and in glanders, in which, also, hæmorrhages occur in mucous membranes. The essential sign of leucæmia is an invariably large (agreeably to the proportion already stated) increase of the white blood corpuscles. The frequent and sudden hæmorrhages of the mucous membranes sometimes cause laymen to confuse leucæmia with anthrax.

Therapeutics.—Treatment is of little use. Besides attending to hygiene, we may give, as in any other affections of the blood, preparations of iron, arsenic (Fowler's solution, 1¼ drs. to 1⅝ oz. for horses in gradually increasing doses), or quinine, which we have found to be a specific remedy against increase in the number of the leucocytes.

Pseudo-leucæmia (*malignant lympho-sarcoma*) is a chronic constitutional disease in which, as in leucæmia, there is a general hyperplasia of the lymph glands of the body, but without increase in the number of the leucocytes. According to our experience, it is by no means rare in dogs. It has been sometimes seen in horses. Nothing positive is known about its relations to other constitutional diseases, or about its origin. Cases of it have, however, been seen to gradually change into true leucæmia. At the beginning of the disease, swelling of the lymph glands is the only morbid change which is apparent, but later on severe anæmia sets in.

HÆMOPHILIA.

Definition.—Hæmophilia is a congenital hæmorrhagic diathesis which manifests itself by persistent and grave hæmorrhages from very slight injuries, or by a tendency to spontaneous and copious bleeding, usually in connection with former injuries. We must here distinguish it from the secondary hæmorrhage which is met with in certain septic and toxic diseases (septicæmia and petechial fever), abnormal condition of the blood (leucæmia and pernicious anæmia), and parasitic diseases (dochmiasis of dogs). The traumatic predisposing causes in hæmophilia are, as a rule, superficial wounds of the skin, incisions in operations for fistulæ,

dilatation of castration wounds, insertion of a seton or rowel, castration, the ulcers of grease, etc. As far as we know, the horse is the only one of our domestic animals which suffers from this disease. Its origin is unknown. In human beings, several cases of well-marked reduction of the thickness of the intima of the arteries, and stenosis of the large vessels with consequent hyperplasia of the vascular system, have been observed. Some authorities believe that the disease is caused by an abnormal plethora of blood in the vessels. Hereditary tendency, even through several generations, may be frequently seen in human hæmophilia.

Symptoms.—As a rule, nothing abnormal can be found in the general health of the animal before the hæmorrhage breaks out; and the disease remains latent for a long time, until an external injury starts it. The blood then rushes persistently, as if from a sponge, out of the wound, no matter how small it may be. In spite of the application of all kinds of means for stopping the bleeding, the hæmorrhage usually lasts for hours, and even days. The blood soon becomes watery and ceases to coagulate; the mucous membranes become anæmic; the frequency of the pulse increases; and the animal becomes debilitated and often bleeds to death in front of the attendant veterinary surgeon. More rarely, the hæmorrhage stops after some hours; but may return at any time. Naturally, the prognosis is always unfavourable.

Therapeutics.—Although we can very seldom master the hæmorrhage by remedial means, success has in a few cases followed their application. Consequently, we may try, first of all, the effect of compression of the wound by ligatures and plugging. If this does not avail, we may employ styptics, such as liquor ferri perchloridi, tannin, alum, creolin, and even the actual cautery. We might also administer internal styptics, for instance, ergot of rye or extract of hydrastis.

SCURVY.

General Remarks.—In human medicine the term scurvy is applied to a hæmorrhagic affection which was formerly very prevalent on board ship, in besieged towns, etc., and even now occurs at times sporadically in prisons and barracks;

Leaving aside premonitory general symptoms, such as debility, weakness, anæmia, and rheumatic pains ; scurvy manifests itself partly by hæmorrhages of the skin, partly by cyanosis, swelling and hæmorrhage of the gums, and ulcerous gingivitis or stomatitis. The other principal symptoms are : hæmorrhages under the skin, between the muscles, on the mucous membranes and in the internal organs ; and complications, such as pneumonia, pleuritis, and arthritis. Scurvy is probably an infective disease. Bad conditions of food and dwelling, unfavourable climate, and particularly the exclusive use of salt meat on board ship, with absence of vegetable diet, were formerly considered to be the chief causes of the disease ; but now appear, with our present state of knowledge, to be only predisposing factors. Accordingly, Garrod's theory that scurvy is caused by absence of the salts of potash has been abandoned.

Pigs and dogs are the only members of our domestic animals in which scurvy has undoubtedly been found. Formerly, any hæmorrhage coming from the gums was erroneously called scurvy. Almost all the cases of scurvy in dogs which have been described in books may be referred to simple ulcerous stomatitis, or perhaps, in a few instances, to septic and toxic affections ; those in horses, to petechial fever or septic processes ; and those in sheep and goats, to anæmia or rachitis. Very few of these descriptions were made with any approach to scientific exactness. Scurvy is evidently rare in dogs ; for we met with only 3 cases of it among 70,000 sick dogs.

Etiology.—In pigs the chief predisposing, if not actually exciting, causes of scurvy are unsanitary conditions of food and dwelling, and want of exercise. Nothing positive is known about the true cause of porcine scurvy ; although Cornevin, Hess, and others consider it to be produced by swine erysipelas. We venture to advance the supposition that scurvy is probably of infective origin in the pig, and also in the dog, which is more exposed than any other domestic animal to those injurious influences which produce scurvy in man. Schneidemükl has observed scurvy in the monkeys of a menagerie, which had been recently landed from a sea voyage.

Symptoms.—The first symptoms are debility and a capricious state of the appetite. The gums assume a violet and, gradually, a discoloured appearance, and bleed from slight provocation. The teeth become loose and finally fall out, and the

animal gets salivated and exhales a very fœtid odour from the mouth. The bristles fall out and show blood on their roots. In dogs and pigs, purple spots and streaks (ecchymoses and vibices), which may change in pigs into deep ulcers, appear on the skin. Swelling of the articulations are met with in a few cases. Besides the spotted hæmorrhages of the gums, we notice in dogs epistaxis, hæmorrhages into the anterior chamber of the eye, and, on ophthalmoscopic examination, hæmorrhages into the retina of the eye. Hæmorrhages of the stomach and intestines may also occur. The animal finally dies from increasing emaciation and debility, which are generally complicated with diarrhœa, if no early amelioration in the external conditions takes place.

The only essential *post-mortem* appearances are hæmorrhages in the skin, subcutis, mucous membranes, brain, and under the serous membranes; and a discoloured condition of the blood, which coagulates with difficulty.

Therapeutics.—The most important remedial means is the removal of unsanitary conditions of feeding and dwelling. As in human cases, we may administer bitters, astringents (radix gentianæ, cortex quercus, and quinine), and preparations of iron. It is also usual to give fruit, acorns, and horse-chestnuts. We have given extract of meat in wine to dogs with apparent success.

Scurvy of Sheep and Lambs.—Lowak states that this disease occurs along with distomatosis and dropsy and that it manifests itself by the teeth becoming very loose, tendency to hæmorrhage and ulceration of the gums, falling out of the teeth, fœtid odour from the mouth and salivation. Other observers have noted the occurrence of continually spreading caries with, finally, perforation of the palate and loosening of the bones of the face, which consequently exhibit an osteoporotic condition. Gips, who has lately made exhaustive researches into the nature of the disease, considers that it has nothing in common with true scurvy; that it is really stomatitis perniciosa of anæmic animals; and that it is due to the gums being easily vulnerable, and to the penetration of particles of food or bacteria into the alveoli of the teeth. The disease, as a general rule, attacks only stall-fed lambs and generally begins on their return from pasture. It spreads epizoötically, particularly among delicate animals of fine-wool breeds. Lambs of ordinary breeds, English sheep which are bred for meat, and older animals are almost entirely free. In spring, at the recommencement of grazing, the disease disappears of itself. It is found most frequently after wet summers and when the animals have been fed on watery food, such as distillers' wash and potatoes. According to Gips, the symptoms are as follows: depression, loss of appetite, frothing from the mouth, congestion and swelling of the gums, and separation of the gums from the maxillary

bones, defects in the epithelium, and ulcers, which conditions are first observed in the canine teeth, and subsequently involve the incisors and finally the molar teeth. Later on, the teeth become loose, alveolar-periostitis sets in, particles of food become wedged into the alveoli, and necrosis of the maxillæ with painful swellings of the lower and upper jaw takes place. Prehension of food and mastication are naturally rendered difficult and painful, and consequently the consumption of food is decreased and the general health suffers. With increased anæmia and emaciation the animal dies from exhaustion. After slaughter, we find excessive anæmia, a watery condition and pale colour of the blood, which has but little reddening effect on white paper dipped into it, gelatinous softening of the adipose tissue, general dropsy, and chronic gastro-intestinal catarrh. As regards treatment, Gips recommends, above all things, a diet easy of digestion and rich in proteine, such as oats, good hay, barley, peas, tares, lupines, and as much green food as possible. The stalls should be roomy, dry, well-ventilated, free from dust, and should be kept at a temperature of from 50° to 55° F. The only medicinal agent to be recommended is iron in the form of sulphate of iron (1½ grs. *pro* lamb), to be given in the drinking water. Gips states that the administration of phosphate of lime is of no use. The teeth may be locally treated with antiseptic and astringent solutions.

GOUT (*Arthritis urica*).

Definition of Gout.—Gout* is caused by an abnormal increase in the amount of uric acid in the blood, in consequence of which uric acid and urates become deposited in the tissues, especially in certain articulations (arthritis urica), and sometimes in the internal organs (visceral gout). It has hitherto been observed,

* The subject of gout is admirably discussed in Dr. Luff's *Gout, its Pathology and Treatment*, from which I have taken the following remarks: Uric acid, $H_2(C_5H_2N_4O_3)$, is formed by the kidneys from urea and probably also from glycocine. In the event of its not being promptly excreted by the kidneys, it unites with the sodium carbonate of the blood and forms sodium quadriurate, which is an unstable and very soluble body. After some time, the sodium quadriurate unites with more of the sodium carbonate of the blood and forms sodium biurate, which is rather insoluble, but is very stable. The following equation shows the conversion of sodium quadriurate into the biurate:

$$2(NaHC_5H_2N_4O_3, H_2C_5H_2N_4O_3) + Na_2CO_3 = 4NaHC_5H_2N_4O_3 + CO_2 + H_2O$$

(sodium quadriurate) (sodium biurate)

As uric acid readily unites with sodium carbonate, it cannot remain in a free state in the blood, which contains a comparatively large amount of sodium carbonate. When the blood becomes more or less loaded with sodium biurate, this compound becomes deposited "almost exclusively in structures belonging to the connective tissue class—in cartilages,

gout can be obtained only by the microscopical or chemical demonstration of urates. If we examine under the microscope the spherical masses of concrete, we shall find that they consist of a regular "felt work" of very fine needles, and that the harder parts are composed of radiating clusters of crystals. The presence of uric acid is chemically proved by placing the concretions on a porcelain dish or watch-glass, moistening them with a few drops of nitric acid, and evaporating the mixture to dryness by means of moderate heat. If the copper-coloured residue be treated with a drop of liquor ammoniæ, it changes into a magnificent purple-red colour, or into purple-blue by the addition of a drop of liquor potassæ. The course of gouty arthritis is very slow.

Therapeutics.—The treatment of gout in poultry is chiefly surgical, and consists in opening the suppurating joints. We may give internally alkalies (a pinch of artificial* Karlsbad salts daily) in the drinking water. These salts, besides other alkalies, in combination with a large amount of water, are given in human medicine for the object of dissolving the uric acid. The newer remedies employed in the treatment of gout in men (piperazin, urotropin, sidonal, etc.), are very expensive, doubtful of efficacy, and may be dispensed with in the cases of animals.

[The researches of Dr. Luff prove that the mineral matter of certain vegetables, such as Brussels sprouts, French beans, winter cabbage, savoy cabbage, turnip tops, celery, and especially spinach, retards the conversion of sodium quadriurate into the biurate, and increases the solubility of the biurate; and that the mineral matter of meat diminishes the solubility of sodium biurate. Hence, these vegetables and many kinds of fruit are an admirable means for relieving sufferers from gout. Strange to say, artificially prepared ash does not produce any good effect in these cases.—TR.]

DIABETES MELLITUS.

General Remarks on the nature of Diabetes Mellitus.—The term "glycosuria," or "mellituria," is applied to a condition in which there is a temporary excretion of grape-sugar in the urine; and that of "diabetes mellitus," to a disease charac-

* Sodii sulphas, *sic.*, 22; potassii sulphas, 1; sodii chloridum, 9; sodii bicarb., 18. 66 grs. of this *sal carolinum factitium* of the German pharmacopœia, dissolved in one quart of water, form a fluid resembling that of the natural spring.

terised by the continued presence of a large amount of sugar in that fluid. Sugar in very minute quantities also occurs, as is well known, in normal urine, especially in that of cattle, sheep and pigs. Besides, we find under normal conditions, a small amount of sugar in the urine of female animals that are bringing up their young, as for instance, in bitches after their milk has dried up, in which case milk sugar (lactosuria), not grape-sugar, is excreted with the urine.

The nature of diabetes mellitus is not fully known. This disease, like albuminuria, is probably only a symptom which is common to several diseases. We may, however, safely say that it is due to some defect in metabolism, in which the sugar, instead of being broken up, is excreted in an unchanged condition from the blood. Consequently, diabetes mellitus is equivalent to glycæmia (increased amount of sugar in the blood). The sugar appears to be derived partly from the hydro-carbons in the food, and partly from increased disintegration of albumen. It seems that the increased sugar-forming activity of the liver plays a great part in the pathogenesis of diabetes. In man, the normal amount of sugar in the blood remains constant at about 0·05 per cent.; but in diabetes mellitus it may rise to 1 per cent.

Occurrence.—Various authors have met with true diabetes mellitus in dogs; Fröhner has seen 7 cases; and Schindelka, Eichhorn, Miller and Fettick, 3 each; Gutzeit and Liénaux, one each. Heiss saw 2 cases of it in horses; and Rueff, Dieckerhoff and De Jong, 1 case each. It seems rarer in other animals. Girotti met with it once in a cow, and Leblanc found it in a monkey. The other cases are either very doubtful or belong to the type of transitory glycosuria.

Theories on Diabetes.—The most important theoretical divisions of diabetes which find acceptance at the present time are as follows :—

1. *Neurogenous diabetes.*—In 1849 Cl. Bernard experimentally produced temporary diabetes in animals by injuring a certain spot at the base of the fourth ventricle of the brain, near the origin of the vagus. Schiff and other physiologists showed that a similar result could be obtained by injuring other parts of the central nervous system, as for instance, the posterior lobe of the vermiform process, by destroying the spinal medulla in the neighbourhood of the origin of the brachial nerves, by dividing the sympathetic, the inferior cervical ganglion, the plexus vertebralis, or the last cervical nerve, by irritating the intercostal nerves, etc. Clinical experience with human beings also shows that sugar is found in the urine after certain injuries to the head, concussion of the brain, and in consequence of pathological changes in the medulla oblongata,

pons Varolii, cervical medulla, etc., as for instance, in cerebro-spinal meningitis, cerebral apoplexy, fractures of the skull, epilepsy, and in mental maladies (psychoses). Pharmacologists have proved that glycosuria can be produced by various brain poisons, such as morphia, amyl nitrite, carbonic oxide, ether, curare, etc. From the foregoing facts we may infer that neurogenous diabetes is a functional neurosis of the central nervous system, especially of the vaso-motor and sympathetic system.

2. *Hepatogenous diabetes* appears to arise from an abnormal action of the functions of the liver. Even Cl. Bernard showed in his diabetes experiments that puncture of the diabetic area in the fourth ventricle of the brain produces a simultaneous hyperæmia of the liver. It is supposed that, either in consequence of hepatic hyperæmia, the glycogen in the liver is passed rapidly and in large quantities into the blood, and that it is changed there into sugar; or that no formation of glycogen takes place in the liver on account of the acceleration of the blood stream, and that the sugar derived from the food is passed directly into the blood.

3. *Pancreatic diabetes* is said to have its origin in changes of the pancreas. According to this theory the pancreatic juice, in the exercise of its normal function, changes the hydro-carbons of the food into products which can be fully oxidised in the blood. When the pancreas becomes affected in such a manner that diabetes is set up, the pancreatic juice is so changed that its action on the hydro-carbons of the food does not proceed beyond the production of grape-sugar, which is excreted with the urine. According to others, in cases of diabetes of pancreatic origin, there is a failure of enzyme which promotes the oxydation of grape-sugar in the blood.

4. *Gastro-enterogenous diabetes*, according to Bouchardat and Cantani, is caused by certain digestive disturbances in the gastro-intestinal canal, owing to the absence of certain ferments in that tube. This theory is founded on the possibility of diet exercising an influence on diabetes.

5. *Hæmatogenous diabetes*, according to Voit, Mialhe, and others, is caused by decreased absorption of oxygen, or diminished alkalinity of the blood, in consequence of which the oxidation of the grape-sugar becomes lessened.

6. *Myogenic diabetes* is said to have its origin in abnormal conditions of muscular activity. Glycogen, which is essentially a nourishing agent for the muscles, and which is completely oxidised under normal conditions, appears, it is said, in considerable quantities as grape-sugar in the blood and urine, when the activity of the muscles has been disturbed. This theory is founded, among other things, on the fact that diabetes mellitus is favourably influenced by muscular exertion and massage.

7. *Constitutional diabetes*, in contrast with accidental diabetes, is supposed to spring from hereditary predisposition; an argument in favour of this view is that glycosuria is hereditary in men.

8. *Symptomatic diabetes* is a concomitant condition of various diseases. A gouty and a syphilitic diabetes are known; also a diabetes of the corpulent (adipose). The diabetes of ingestion (phlorizin)* and the

[* Phlorizin is a glucoside, the consumption of which causes diabetes. Villaret states that it is found in the bark of the roots of apple trees, pear trees, plum trees, and cherry trees.—TR.]

diabetes of confinement in dogs and cats, are other isolated forms of diabetes mellitus. Finally, we have in various quarters the opinion that diabetes is neither due to a single cause, nor is of a single type, but is referable to a complex of various processes in the body. According to Stokvis, the morbid process in diabetes consists in the disturbance of several functions ; for instance, the formation of sugar in the intestinal canal, the formation of sugar and glycogen in the liver, and the consumption of sugar in the muscles.

Symptoms.—1. In *dogs* the first symptoms of diabetes mellitus are dulness, weakness, inability to bear fatigue, and gradual emaciation. If the disease be not too far advanced, the appetite is usually remarkably voracious and the thirst excessive. The urine is very abundant and is more frequently voided than in health. Its specific gravity is generally increased (1·040 to 1·060). The amount of grape-sugar in the urine varies, and may be as high as 12 per cent., which was the proportion found by Haltenhoff in one case. We have observed in dogs, respectively, 4, 5, 7, and 8 per cent. Wolff met with cases showing from 7 to 8 per cent. In such severe instances, the presence of sugar in the urine may be recognised by the sweet taste of that fluid. A grey cataract (cataracta diabetica) may develop in both eyes, which may finally become completely blind (Wolff, Haltenhoff and Ourselves). Schulz and Strübing found bilateral detachment of the retina in a dog which was suffering from mellituria produced experimentally by feeding on sugar. The animals are often affected with vomiting, cough, diarrhœa, hæmorrhages of the mucous membranes (*Thiernesse*), and ulcers of the cornea. The debility becomes excessive towards the end of the disease.

The course of the disease is always slow and extends over several months. Although recoveries may occur, as we have seen in our own practice, the prognosis is on the whole very unfavourable. Death sometimes takes place very rapidly with stupor (coma diabeticum). Post-mortem examination displays, as a rule, no characteristic alterations, beyond a pronounced degree of fatty infiltration of the liver (Fröhner, Eber, Schindelka, Wolff) ; atrophy of the pancreas has also been found sometimes (Eber, Liénaux, Almy).

2. Heiss described in detail cases of diabetes mellitus in two Belgian cart *horses*, which were respectively 10 and 11 years old. Both were in the same stable and in the same team. The disease showed itself by debility, depression, atony of the muscles, and general disturbance of health, which was chiefly

manifested by the unthriftiness of the animals' coats. The consumption of water rose from 3 to 5 times the normal quantity, and the appetite also became greater than usual. The increased activity of both these functions lasted until death. The nauseous, sickly-smelling urine gave, by Trommer's test, a large, orange-coloured precipitate of suboxide of copper, and was found to contain on an average 3.75 per cent. of grape-sugar. Both horses developed cataracta diabetica and ulcus corneæ in the fifth week of the disease, and died from excessive emaciation and exhaustion 2 months after they had been taken ill. The most important pathological changes found on *post-mortem* were a peculiar clay-coloured appearance of the liver and enlargement of that organ.

The case described by Rueff was that of a ten-year-old gelding which had been unsuccessfully treated for five weeks, and which was greatly reduced in condition and strength, though the appetite was good and the consumption of water greater than usual. The urine was abnormally copious, and on being examined by a chemist showed a specific gravity of 1.052, and contained 5.85 per cent. of sugar. Dieckerhoff described a similar case (0.2 to 0.6 per cent. of sugar in the urine). In the case observed by De Jong, there was polyuria, with high specific gravity (1.060), and much contained sugar.

Girotti observed, in *cattle*, polyuria (22 gallons per diem) and great thirst (33 gallons of water per diem); wasting, in spite of liberal ingestion of food; and presence of sugar in the urine.

Diagnosis.—We may suspect diabetes mellitus when we find that, without apparent cause, the animal becomes gradually debilitated and emaciated, although the appetite continues good or even increased; that the amount of water consumed and urine excreted is increased, and that cataract becomes developed. A positive diagnosis cannot be made without testing the urine for grape-sugar. The easiest test is that of Trommer, in which, after placing some of the urine in a test tube, we add a 15 per cent. solution of caustic potash until a strong alkaline reaction is produced, and then add by drops a solution of sulphate of copper, until the fluid shows a clear deep blue colour (formation of copper hydrate). If we then heat the test tube, the dark blue colour will gradually change into the opaque yellow or orange of the sub-oxide, which will be precipitated if the urine contains more than 5 per cent. of grape-sugar. The quantitative estimation of sugar with Fehling's solution is based on the same pro-

cess of reduction.* Böttger's test depends on the reduction of the sub-nitrate of bismuth into black metallic bismuth by boiling in an alkaline solution ; Moore's potash-test gives a deep brown colour to the urine, to which liquor potassæ has been added, after the upper layer of the fluid has been carefully heated. Grape-sugar in the urine can be demonstrated by the polariscope (apparatus of Mitscherlich) as it turns to the right the plane of polarisation; hence the name "dextrose." By this apparatus we may also very easily obtain a quantitative analysis of sugar. A surer method is that of fermentation, in which the presence of yeast causes the sugar to be broken up into carbonic acid and alcohol (introduction of a quantity of urine with some yeast into a granulated Eichhorn fermentation tube, leaving it for 24 hours in a warm place). It is advisable always to control the Trommer's test with that of the fermentation process; as recent investigations have shown that besides the ordinary diabetes due to the presence of grape-sugar, there is a glycosuria which consists of the presence in the urine of non-fermentable pentoses (pentosuria).

* Mr. W. H. Willcox, B.Sc., A.I.C., has kindly sent me the following explanation of the reactions in these two tests :—

In Trommer's fluid we have in the first instance :

$$CuSO_4 + 2NaOH = Cu(OH)_2 + Na_2SO_4.$$

If no sugar be present in this cold solution, the hydrate will form a *greenish blue* precipitate, which will then remain unchanged ; but which, on being boiled, will turn brown, owing to the formation of CuO. The reaction is as follows :

$$\underset{\text{(greenish blue)}}{Cu(OH)_2} = \underset{\text{(brownish black)}}{CuO} + H_2O.$$

If, however, sugar be present in sufficient quantity in the cold solution, it will prevent the precipitation of the $Cu(OH)_2$, which will accordingly give a greenish-blue colour to the liquid ; and it will, under the action of heat, change the hydrate into Cu_2O (cuprous oxide or suboxide) by the abstraction of oxygen : becoming itself converted into gluconic acid ($C_6H_{12}O_7$). Thus :

$$2Cu(OH)_2 + C_6H_{12}O_6 = Cu_2O + 2H_2O + C_6H_{12}O_7.$$

Fehling's test is based on the fact that the presence of sodium-potassium tartarate causes the solution of the greenish-blue precipitate $Cu(OH)_2$, which then gives a *deep blue* colour to the liquid. Hence, Fehling's fluid (a mixture of $CuSO_4$, NaOH, and $NaKC_4H_4O_6$) is practically a solution of $Cu(OH)_2$; and when it is boiled it gives exactly the same reaction as that of Trommer, namely, the formation of the red precipitate of cuprous oxide.

Fehling's test is considered the more elegant one of the two ; because it does not entail delay by the precipitation of the hydrate which is held in solution.

Fehling's and Trommer's tests are so delicate that they will easily indicate the presence of a solution of grape sugar of 1 in 5,000.—Tr.

Therapeutics.—The treatment of diabetes mellitus chiefly consists in reducing as much as possible the supply of hydrocarbons in the food. Sugar-formers, the most important of which are starch, sugar itself and glue-yielding substances, should be replaced by albumen and fat. We have proved in two cases that an exclusive meat diet is best for dogs. In human medicine, alkalies, especially Karlsbad water and salt, opium, carbolic acid and salicylate of soda are administered, and saccharine is used so that the food may not be entirely deprived of a sweet-tasting element.

DIABETES INSIPIDUS (*Polyuria*).

Definition.—In human medicine the term diabetes insipidus is applied to a disease which is probably due to nervous disturbance, and is characterised by the excretion of an abnormally large quantity of non-saccharine (*insipidus, i.e.*, tasteless) and very watery urine. Nothing is known respecting its origin, although it appears probable that it has some connection with diabetes mellitus, as it may change into it. It is stated that diabetes insipidus may be produced in the ordinary course of events by injuries of the brain and various acute morbid processes; and experimentally by injury of the fourth ventricle in the neighbourhood of the centre of the vagus.

It has not been positively proved that animals suffer from a disease which is identical with diabetes insipidus in men. In veterinary medicine it has hitherto been the custom to designate every polyuria as diabetes. Polyuria differs from diabetes insipidus by the fact that it is merely a symptom, and often only a temporary symptom, of morbid conditions produced by the absorption of exuded material during the course and towards the end of many acute diseases, especially contagious equine pleuro-pneumonia, strangles, pharyngitis, diseases of the medulla oblongata and of the cerebellum, many diseases of the kidneys, especially chronic interstitial nephritis (the so-called contracted kidneys), and hyperæmia of the kidneys; and during chronic diseases such as leucæmia, glanders, and tuberculosis. Severe polyuria sets in after the administration of certain poisons, such as

cynanchum vincetoxicum, which sometimes gives rise to enzoötic poisoning in sheep. The principal cause of the ordinary kind of polyuria, which we often find as an enzoötic among horses in badly-managed stables, is, as we all know, the consumption of musty oats. Damp oats having been kept for a considerable time without being turned over, as for instance on board ship, ferment, probably, from the action of mould fungi. Dammann observed that musty peas also produce this polyuria. This toxic polyuria has just as little in common with true diabetes insipidus as the polyuria of contracted kidneys. The nearest approach in horses to human diabetes insipidus is found in those cases of polyuria which have no apparent cause, and which exhibit no characteristic changes on *post-mortem* examination. Stockfleth states that he found true diabetes in a horse after a chill; and Perrin, after a kick on the region of the liver. Schindelka describes, in a dog, one case which was distinguished by its rapid progress, and by complications (cataract and abscess) peculiar to diabetes mellitus.

Symptoms.—Although the equine form of polyuria due to musty fodder differs as to cause from human diabetes insipidus, it presents similar symptoms. The digestion becomes impaired a few days after the consumption of the musty oats, and colicky pains may also ensue. Attention is soon attracted to the abnormal frequency of the act of micturition, and to the large quantity of excreted urine, which in severe cases may amount, daily, to 5 and even to 11 gallons. The urine is pale, very watery, poor in solid constituents, and of the low specific gravity of from 1·001 to 1·015. It contains neither albumen nor sugar, and often has an acid reaction, probably on account of simultaneous intestinal disturbances. The increased secretion of urine is accompanied by increase of thirst, which often becomes very excessive. Horses have been known to consume in one day from 17 to 22 gallons of water. The temperature is usually normal, but may become considerably heightened during severe gastric attacks. Marked debility is generally present.

In the majority of cases the symptoms just described disappear after the supply of the injurious oats has been stopped. It seems that neither recovery nor improvement is possible if the system has been under the influence of the toxic matter for a long time. In such cases the animal becomes more and more emaciated, and finally dies from cachexia after the disease has lasted for months or even for years.

Therapeutics.—Removal of the cause, namely, stopping the injurious food, is the best treatment. If this cannot be done the oats may be improved by washing, cleaning, and frequent shifting.* It has long been the custom to administer various internal astringents, such as sugar of lead, sulphate of iron, etc.; but we are unable to express our opinion on their value. Karlsbad salt, opium (as a thirst-quenching agent), valerian root and ergot of rye, are said to be of use in diabetes insipidus of men, and may be tried in chronic cases of equine polyuria. Almy claims the cure of diabetes in a dog with potassium bromide; Hasse, in a horse, with extractum hydrastis; Röder recommends tannopin (¼ oz. two or three times a day).

OBESITY.

Definition.—Obesity is an abnormally large deposition of fat in the body, especially in the subcutaneous connective tissue. We endeavour to produce it physiologically in animals which are destined for slaughter, and in individuals of certain breeds. Pathologically it occurs chiefly in animals that are kept for breeding, and in dogs, which are often treated for it by veterinary surgeons. Its treatment in animals is of far less pathological importance than in human beings.

Etiology.—The principal causes of obesity are: abundant consumption of fat-forming food and want of exercise. Many dogs, especially lap-dogs and house-dogs, and stud animals, such as bulls and boars which remain permanently in their stalls, and which are too highly fed, often become very obese. The adipose tissue is formed from the albumen as well as from the fat contained in the food. The hydro-carbons of the food participate only indirectly in this process, by diminishing the loss of albumen in the body. Hereditary predisposition may be so strong in certain breeds of pigs, cattle, sheep, and possibly of dogs, as to induce obesity independently of the factors just mentioned. Anæmia is another predisposing cause of obesity,

* Heat, applied for instance by kiln-drying, boiling, steaming, or parching (as is done in India), is the best means for destroying the pathogenic fungi. Curtailment of the supply of the horse's drinking water would not only be useless cruelty, but would also have the bad effect of checking the excretion of morbid material from the system.—Tr.

as we may learn from the success of the empirical practice of bleeding animals which are intended to be fattened for slaughter.

Symptoms.—Fat animals appear of a more rounded outline of body, and statelier appearance. The panniculus adiposus is greatly developed, so that the skin often forms broad folds and ridges. Many animals, especially pug-dogs, become abnormally stout. With increase of the well-known signs of obesity, there is loss of functional power and vivacity. They appear heavy and lazy, are easily fatigued; the appetite becomes capricious, digestion disturbed and the procreative faculty impaired or even destroyed. In cases of long continued and excessive obesity, dyspnœa becomes complicated with acceleration of the pulse, palpitation of the heart, and disturbances of the circulation, such as hypertrophy of the heart, fatty infiltration of the heart, and finally fatty degeneration of the heart.

Therapeutics.—Obesity may be treated by giving less food, by reducing the quantity of some of the component parts of the food, or by increasing the decomposition of the adipose tissue.

1. Decrease in the total amount of the food is the simplest and most reliable remedial means. In our clinic we have always succeeded in making fat dogs lean in a comparatively short time by a simple reduction of the diet. This plan is not always practicable; for owners cannot be relied on to enforce the necessary regimen.

2. A decrease or withdrawal of food constituents, such as fat and hydro-carbons, which tend to form adipose tissue, has been tried in man with various modifications, the oldest of which is that of Banting. His "cure" consisted in the almost entire elimination of hydro-carbons and fat, so that the diet was practically restricted to lean meat. Good results may be expected from this method, if it be judiciously applied for moderate periods of time. Its too strict and long continued employment gives rise to aversion from albuminous food, and indigestion, followed by catarrh of the stomach and intestines, general weakness, and exhaustion. On account of the absence from the food of fat and hydro-carbons, which, when digested, protect the albumen of the tissues from disintegration, the body consumes a portion of its own albumen which, in any case,

is not very abundant in obesity. Elbstein's method excludes hydro-carbons, except fat, which may be taken in small quantities. It appears to reduce the feeling of hunger and thirst, and to favour the deposition of albumen in the body.

[In man the only dietetic method for checking obesity which can as a rule be permanently adopted with good results, is the exclusion of starch (bread, biscuits, cakes, pastry, macaroni, vermicelli, rice, sago, cornflour, tapioca, arrowroot, etc.), and a liberal supply of those fruits (apples, pears, grapes, pine-apples, oranges, strawberries, raspberries, plums, peaches, greengages, cherries, etc.) and vegetables (cabbages, cauliflowers, tomatoes, celery, spinach, etc.) which are poor in starch. TR.]

3. Fat is best reduced by exercise, which accelerates the circulation, increases metabolism, and strengthens the action of the heart. The benefits of exercise are well shown in the training of horses.

4. Deprivation of water, agreeably to the method introduced by Œrtel, relieves the heart of work by decreasing the amount of blood, and consequently increases metabolism. This method may be utilised in the severe affections of the heart which occur during the later stages of obesity. It can be combined with exercise.

[This system has the very serious disadvantage of tending to check metabolism and to produce constipation. Consequently, its application is often followed by rheumatic affections brought on by undue retention of effete and deleterious material in the system, and by digestive disturbance. TR.]

5. The old remedies against obesity, such as salts of bromine, alkalies, aperients and diaphoretics, have fallen more or less into disrepute, and have given way to the purely dietetic method of treatment; because they operate too violently, and when successful, they obtain their effect partly by the artificial production of a catarrh of the stomach or intestines. The alkalies which increase metabolism, such as Karlsbad salt, are the least injurious medical agents for the reduction of obesity. On the other hand, the organo-therapeutic preparations which have recently been used for human beings, derived from the thyroid gland (iodothyrin, thyreoidin) may also be used, with caution, in the treatment of the domestic animals (1½ to 3 grains per day for a dog).

SARCOMATOSIS, CARCINOMATOSIS, AND SCROFULA.

THE formation of sarcoma and carcinoma comes into the province of medicine when they occur in the internal organs, such as the stomach, liver, intestines, kidneys, uterus, lungs, brain, etc., or when they spread through the whole body. Sarcomatosis and carcinomatosis often assume in horses, and especially in dogs, the aspect of chronic peritonitis or pleuritis with abundant serous or hæmorrhagic exudations; as extensive new-growths of a sarcomatous or cancerous nature may become localised in the pleuræ and peritoneum. The sarcomata of the abomasum give rise in cattle to chronic indigestion and loss of condition. With the frequent carcinomata of the mammæ of bitches, we sometimes observe metastatic formations in the internal organs, such as the liver, uterus (with a fœtid, sanious discharge from the vagina) and lungs (with symptoms of pulmonary consumption). Periosteal generalised sarcomata in dogs may give the appearance of articular rheumatism; sarcomata of the lungs and carcinomata in cattle and horses, of tuberculosis; sarcomata of lymph glands in horses, of glanders or of leucæmia; etc. If such processes last a considerable time the animals become greatly emaciated. Ulcerations of the new growths are complicated with continued septic fever.

Treatment being out of the question, we can only attempt to strengthen the system by good feeding and to check the increase of the cancer cells by small doses of arsenic in the form of Fowler's solution.

SCROFULA.

Scrofula is an historical collective definition, and not an independent disease. Whilst in the earlier text-books it played rather an important part, it has in recent years been wholly removed from the list of primary diseases and been regarded as a concomitant of the most various pathological changes. According to old clinical text-books, the principal symptoms are swelling of the external and internal lymph glands with suppuration, caseation and induration; catarrh of the mucous membranes; cutaneous eruption; gradual loss of condition; and finally cachexia. The majority of the reputed cases of scrofula are evidently those of tuberculosis, a fact to which Spinola called attention even in his time. Other cases were those of rachitis (principally in pigs), swine fever, parasites in the lungs (strongylus paradoxus in pigs), pyæmic polyarthritis in foals, and metastatic strangles (strangles was formerly called "*scrofula equorum*"). In aged animals it was probably mistaken for cases of leucæmia, pernicious anæmia, and chronic intestinal catarrh. Cachectic diseases of an unknown kind were generally classed as scrofula. In foals, too, scrofulosis was commonly designated by the term *darrsucht* (wasting).

CHAPTER VIII.

INFECTIVE (OR INFECTIOUS) DISEASES AND PLAGUES IN THE STRICTER SENSE OF THE TERMS.

SEPTICÆMIA AND PYÆMIA.

I.—SEPTICÆMIA.

Nature and Causation.—By the terms septicæmia (sephthæmia, sepsis, blood-poisoning, putrid fever) is denoted a grave disease originating in wound-infection, from the introduction of bacteria and their products of decomposition into the blood; which, in contra-distinction to pyæmia, usually display no local affections or lesions (metastases) in either the internal or external organs, but are chiefly distinguished by general changes; especially by tumefaction of the spleen and parenchymatous inflammation of the liver, kidneys, heart and other internal organs. Moreover, it often happens that septicæmia is combined with pyæmia (septicopyæmia, pyosepticæmia).

With regard to its etiology, two principal types of septicæmia are distinguished, one being generated by schizomycetes and the other by dissolved chemical poisonous matter (toxins). That which originates from schizomycetes is distinguished as bacterial septicæmia, bacteriæmia or septic infection, and that which is dependent upon toxins as septic intoxication. The first is transmissible to other animals by blood inoculation, while the blood of the latter is not infective. Between these two transitions and combinations occur mixed infection. When the origin of the septicæmia cannot be discovered, it is said to be cryptogenetic septicæmia.

1. Bacterial septicæmia is produced by various schizomycetes—cocci as well as bacilli. On this account, we can distinguish between cocco-septicæmia and bacillo-septicæmia. Septicæmia is produced more especially by cocci—the strepto-

coccus septicus and the micrococcus tetragenus ; but the cocci which are present in pyæmia, namely streptococcus pyogenes and staphylococcus aureus, can also give rise to septicæmia. Koch proved experimentally that the respective bacilli of the septicæmia of mice and of the septicæmia of rabbits are pathogenic ; to this class also belong the bacillus coli and the bacillus enteritidis. In contrast to these simple ones, the following may be classed as specific septicæmia: malignant œdema, splenic fever, quarter-ill, bacterial erysipelas, swine-fever, the septic variety of so-called paralysis of foals, the septicæmia of calves, septicæmia hæmorrhagica (deer-plague), the cholera of fowls and fowl-plague.

2. Septic intoxication consists in the absorption of poisonous products of the metabolism of bacteria. These substances, which are known as toxins, ptomaïns, putrefactive poison, cadaveric poison, and flesh poison, are chemically very different bodies (toxalbumins, albumoses, organic bases—especially amin bases and nuclein bases—fatty acids and aromatic products). The source of these toxins is usually derived either from a centre of putrefaction situated on the external surface of the body, or one in the uterus, intestine, lungs, or liver. As a rule it is the above-named schizomycetes, which have settled in dirty wounds, or in retained placentæ, which are undergoing putrefaction (puerperal septicæmia), or in putrefactive centres in the intestine or lungs, that are present, the decomposition-products of which have been there absorbed. Non-pathogenic fungi, especially the bacteria of putrefaction, can produce very strong chemical poisons by the septic decomposition of the animal tissues, after these fungi have penetrated into the purulent or serous foci. The absorption of these products of metabolism gives rise to general intoxication. This form of septicæmia, which is caused by putrefactive fungi (saprophytes), is called "sapræmia." Specially dangerous in this connection are the proteus vulgaris (bacillus proteus), and the nearly related bacillus cellulæformans (flesh-poison). The entrance of the saprophytes, even into the blood, was formerly denied to be a cause of septicæmia ; but, according to more recent observations, it appears that this may sometimes result.

Anatomical Conditions.—In the *post-mortem* examination of animals which have died of septicæmia, we find in the first place a tar-like, varnish-coloured aspect of the blood ; which, as well as the solid constituents of the body, shows a

striking tendency to decomposition. In the microscopic examination of the blood in cases of bacterial septicæmia, the corresponding schizomycetes are found, which have led to destruction of the white and red blood-corpuscles. The white corpuscles, especially, are converted by the multitudinous crowding of the bacteria contained in them, into what appear to be bacterial colonies. As a result of parenchymatous disease of the walls of the vessels, hæmorrhages occur in the mucous membranes; also beneath the serous membranes, especially the endocardium; into the mesentery and the omentum; and also into the substance of the kidneys, spleen and liver.

Spleen, liver and kidneys display, as a rule, parenchymatous swelling; the heart muscle, and sometimes the muscle of the skeleton, appear loam-coloured, as if boiled. In many cases there occurs, besides an ulcerative endocarditis, a catarrhal, hæmorrhagic and even ulcerative enteritis; also a parenchymatous and hæmorrhagic nephritis. In very acute cases of septicæmia, all these changes are slightly expressed; especially in the toxic form, in which, as in cases of poisoning, the results of dissection may be purely negative. (This should be a warning with regard to the inspection of meat!)

Symptoms.—Septicæmia is, of all infective wound-diseases, the most important and the most common. It is found in the horse, especially after traumatic, ichoro-purulent inflammations of the joints (fetlock and hock-joints), and of the tendon-sheaths; also in cases of severe septic, subfascial, and intermuscular phlegmonous inflammations. In cattle and in dogs, it frequently occurs as a sequel of parturition (puerperal septicæmia). *Pigs are, on the other hand, found by experience* to be but slightly susceptible to it (castration). The local changes vary according to the starting-point of the disease.

1. In the septicæmia of *the horse* there is found a septic inflammation of a phlegmonous type, around the wound. Sometimes, however, there is an absence of marked changes at the seat of injury (hyper-acute cases). The general disease often begins with severe febrile symptoms, a temperature up to 104° Fahr. or higher, sometimes accompanied by rigors; a very frequent, small, and at last wholly imperceptible pulse, with great cardiac weakness. In many forms of septicæmia, however, a pronounced rise of temperature may be absent. In addition, there is severe general disturbance: diminished appetite, though sometimes the animals will eat till

shortly before death; extreme lassitude and weakness; pronounced implication of the sensorium; muscular twitchings; copious and persistent sweatings; sometimes also paralysis of the hinder parts; a dirty-red or icteric colouration of the mucous membranes, accompanied by formation of ecchymotic patches; discoloured and albuminous urine; also, towards the end, profuse diarrhœa, accompanied sometimes by colic. The duration varies greatly; the disease may end fatally within the course of a day; but it usually lasts several days, and has sometimes extended over weeks (inflammation of fetlock joint).

2. Puerperal septicæmia (the septic variety of puerperal fever), is that which most frequently occurs in *cattle*. Here we may indicate, clinically, the distinction of two forms of septicæmia: infection and intoxication. The puerperal infection manifests itself either as a puerperal phlegmonous inflammation, or as a septic metritis (fever, sense of oppression, groaning, tenderness of the abdomen on pressure, a fœtid, chocolate-coloured uterine discharge, croupous-diphtheritic changes displayed on dissection); or, as an acute puerperal septicæmia, which was formerly included with the general phenomena of sepsis (sudden cessation of eating and secretion of milk, high fever, yellowness of mucous membranes, prostration); and which, as a rule, ends fatally after from one to three days, while it frequently leaves but slight changes, and these of a general kind, which can be discovered on dissection. Puerperal intoxication passes either under the form of parturition-paresis (paralytic calf-fever, auto-intoxication) or some slighter symptoms of disease (weakness, gastric derangements, normal temperature). Retention of the placenta more especially displays a relatively mild form of sapræmia; in rare cases it causes sudden development of paralysis, and death after a few hours (De Bruin).

Treatment.—As in wound-fever, so also in septicæmia, the local antiseptic treatment of the wound is of the gravest importance. The employment of strong disinfectants, the thorough removal of stagnant wound-secretions, the careful drainage of the wound, the earliest possible incision of areas of fluctuation, the antiseptic douching of the puerperal uterus, the manual detachment of any retained membranes, are the most effective remedies. Internal treatment with antipyretic agents is of but secondary importance; those which are most highly

striking tendency to decomposition. In the microscopic examination of the blood in cases of bacterial septicæmia, the corresponding schizomycetes are found, which have led to destruction of the white and red blood-corpuscles. The white corpuscles, especially, are converted by the multitudinous crowding of the bacteria contained in them, into what appear to be bacterial colonies. As a result of parenchymatous disease of the walls of the vessels, hæmorrhages occur in the mucous membranes; also beneath the serous membranes, especially the endocardium; into the mesentery and the omentum; and also into the substance of the kidneys, spleen and liver.

Spleen, liver and kidneys display, as a rule, parenchymatous swelling; the heart muscle, and sometimes the muscle of the skeleton, appear loam-coloured, as if boiled. In many cases there occurs, besides an ulcerative endocarditis, a catarrhal, hæmorrhagic and even ulcerative enteritis; also a parenchymatous and hæmorrhagic nephritis. In very acute cases of septicæmia, all these changes are slightly expressed; especially in the toxic form, in which, as in cases of poisoning, the results of dissection may be purely negative. (This should be a warning with regard to the inspection of meat!)

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ngitis, lymphadenitis, and formation of metastases). Some-imes the original source of the pyæmia remains obscure (crypto-enetic pyæmia).

Besides, in many cases a distinction between pyæmia and epticæmia is not possible. When a combination of the two onditions exists, it is spoken of as pyo-septicæmia.

Anatomical Conditions.—In cases of pyæmia, the characteristic anatomical changes consist, besides the pus cocci in the blood, of more or less numerous purulent foci (metastatic processes) in both internal and external organs—lungs, liver, spleen, kidneys, brain, heart, muscles, joints, tendon-sheaths, etc. In paralysis of foals, especially, the multiple suppurative inflammation of the synovial membranes of the articulation appears in the foreground (polyarthritis pyæmica). We meet with suppuration of the serous membranes—such as the peritoneum, pleuræ, meninges; and in the eye in the form, for instance, of purulent choroiditis and panophthalmia. We may observe, too, as in septicæmia, ulcerous endocarditis and numerous circumscribed hæmorrhages in the serous membranes, skin, eyes, muscles, etc. There may also be anatomical changes peculiar to septicæmia (septico-pyæmia). The local changes also are, in some cases, very characteristic. We find, as the result of the emigration, numerous bacteria in the walls of the veins around the wound (*e.g.*, umbilical); a purulent inflammation of the venous walls, with purulent decomposition of the contained thrombus (suppurative thrombophlebitis), which is the source of the purulent embolic foci in the interior of the body, as well as of the numerous bacteria (micrococci) in the blood and internal organs.

Symptoms.—Pyæmic wound-infection begins with fever of a variable, frequently intermittent, and very irregular form; sometimes, also, with rigors. Then are developed the evidences of a pulmonary inflammation, or the symptoms of abscesses in the liver, kidneys and brain; pyæmic polyarthritis, tendovaginitis, pleuritis, meningitis, etc. Sometimes multiple subcutaneous purulent foci are observed, which arise in the form of phlegmonous swellings in various parts of the body—often quite suddenly, and frequently in large numbers. The duration of pyæmia is usually more prolonged than that of septicæmia; it generally lasts from several days up to some weeks, according to the origin and diffusion of metastatic formations. Sometimes

a chronic pyæmia with great emaciation of the animals is developed. Recoveries are somewhat more frequent in pyæmia than in septicæmia, but are, generally speaking, rare, and the stage of convalescence is always very prolonged.

Puerperal pyæmia (the pyæmic variety of puerperal fever) in cattle (it seldom occurs in horses), is specially characterised by febrile polyarthritis of the fetlock and carpal joints, mastitis, osteomyelitis, tendovaginitis of the flexor tendons, and chronic parametritis (multiple abscesses in the connective tissue of the pelvic cavity, chronic emaciation).

The pyæmia of strangles (so-called wandering or erratic strangles), is specially distinguished by formation of abscesses in the various lymphatic glands of the body (upper, middle and lower cervical glands ; axillary, bronchial, mesenteric, pelvic, lumbar and popliteal glands) ; also by the formation of abscesses in the brain, spinal cord, omentum, udder, kidneys, pancreas, orbita, etc.

Treatment.—This consists, as in septicæmia, chiefly of thorough disinfection, draining and incision, of antipyretic remedies ; quinine should be used tentatively on account of its specific effects. Internally the employment of argentum colloidale has been recommended (intravenous injection).

Septicæmia of Calves.—Thomassen has observed a new variety of septicæmia which appeared in epidemic form among calves in the neighbourhood of Utrecht. It usually showed itself within from five to eight days after birth, and its course was accompanied by hæmorrhagic nephritis, cystitis and urethritis. He has cultivated a specific bacillus from the blood in this affection, and has obtained results from its inoculation on healthy calves. According to Nocard, it is produced by a variety of bacillus coli (Zschokke).

Septicæmia of Fowls.—Legrain and Jacquot have described a form of pyæmia which is peculiar to fowl, and which frequently occurs in Lorraine, where it causes great loss. It is characterised by extensive abscesses on the head and neck, and especially about the eyes. If these abscesses are neglected, they may produce a chronic septicæmia, which leads to severe and progressive emaciation that ends in death ; but if treated surgically, they rapidly heal. The disease can be transmitted to pigeons, which die in from 15 to 30 hours, and also to mice. It is caused by short, stout bacilli which have rounded and shining ends. These bacilli are from 1 to 2 μ long, and cannot be stained by Gram's method. Their cultures, according to Legrain and Jacquot, resemble those of the bacillus septicus aerigenus of Nicolaïer, the bacillus saprogenus of Rosenbach, and the bacillus pyogenes fœtidus of Passet, which these authors consider to be identical with each other. The poultry infect themselves on the dunghills in which they seek their food.

Norgaard found a streptococcus which was the cause of an apoplectiform septicæmia in fowl (with a mortality of 100 per cent.); a similar disorder has been described by Mazza and Rabieux.

Septicæmia of Geese.—Sakharoff describes a typhus-like summer epidemic of Russian geese, which manifests itself by high fever, diarrhœa, and emaciation, and runs a fatal course in about a week. He found in the blood of living animals, spirochæta anserina, which resemble the spirilla of relapsing fever, and which he successfully transmitted to geese. McFadyean found in an epidemic of the septicæmia of geese, to which 150 were victims, (an intestinal inflammation—the duration of the disease being from half an hour to a day), that the cause was a cylindrical bacterium resembling the bacillus of erysipelas in size and form. Inoculation experiments made on geese gave positive results.

Septicæmia of Rabbits.—The disease described by Davaine, Koch and Gaffky is produced by the bacillus cuniculisepticus (Bacterium cuniculicidum), which, according to Kitt and Hueppe, is identical with the bacterium of the cholera of fowls.

MALIGNANT ŒDEMA.

Etiology.—Koch's malignant œdema is a specific wound-infection disease, in the form of a specific phlegmonous inflammation, or sero-hæmorrhagic infiltration of the connective tissue with formation of gas, which is produced by small spore-forming bacilli (bacilli of œdema, bacillus œdematis maligni). These are distributed in nature with an excessive profusion (ubiquitous schizomycetes), and are specially to be found in the upper strata of the soil, and in the hay-dust and sweepings as well as in the saliva and excrement of graminivorous animals. A rabbit will die in from 24 to 36 hours from malignant œdema, if a small quantity of ordinary garden-soil is placed under its skin. The infection takes place in consequence of contamination of wounds of the skin by earth, fæces, hay-dust, etc.; but only when the bacillus of œdema gets into the subcutaneous connective tissue. The introduction into the blood of such infective material is comparatively innocuous; apparently because the oxygen of the blood kills these anærobes. Inoculation from the cutis is also unsuccessful. The process of granulation hinders the entrance of the bacilli into wounds. Before the bacilli of œdema can penetrate into the subcutaneous and submucous connective tissue, they must be, to a certain extent, prepared for their reception; as for instance by the presence of such nutrient fluids as serum, lymph, and blood.

The site of the infection must also be as free as possible from blood which contains oxygen. Consequently, the more the circulation is interrupted on the site of the infection, the better will these bacilli develop on it. According to more recent researches, (Besson) pure spores of the bacillus of œdema can undergo no further growth in the healthy tissues of living animals (phagocytosis). Their development is much rather dependent on association with other bacteria ("favourable microbes"), especially with staphylococci. These facts explain that, as in tetanus, in spite of the frequency of the presence of the bacillus of œdema (ubiquity), cases of malignant œdema in animals are rare. The occasions of infection are usually afforded by injuries of the tongue; subcutaneous infections; scratches; punctured wounds; injuries of the valva, vagina and uterus; operations with unclean instruments; castrations; docking; shearing (of sheep); etc. Chauveau states that an attack confers subsequent immunity.

The bacilli of œdema closely resemble in size and shape those of anthrax, except that they are more slender, are under certain circumstances motile, and have rounded ends. After the death of infected animals, the bacilli grow very long, and form partly stiff or slightly-bent and loop-like threads on which divisions can be recognised, so that they present the appearance of bacilli attached to one another. Later on, the spores develop from these threads. After 24 hours, the bacilli of œdema are found in great numbers in the blood (especially in that of the portal vein) of animals which have died from suffocation, if the cadaver has been kept at a temperature of 100·4°F. For this reason they may be found, as a rule, within from 12 to 24 hours in the blood of the liver and spleen, and afterwards throughout the entire blood of the body in the cadavers of large domestic animals which have died of conditions associated with dyspnœa; especially in cases of colic, when the bodies have remained a considerable time unopened in presence of a rather high external temperature (so-called cadaveric bacilli).

According to the investigations of Kitt, malignant œdema may be easily transmitted by inoculation to all our domestic animals (goats, calves, sheep, dogs, horses, guinea-pigs, fowl, and pigeons).

Bacteriology.—The bacilli of malignant œdema are spore-forming rods, anærobic and generally very motile. They liquefy gelatine, can be stained with Gram's solution, are from 3 to 5 μ long, and 1 μ broad, and are consequently about four times as long as they are broad.

Several rods usually form pseudo-threads of from 10 to 40 μ in length. Although the bacilli are at once destroyed by oxygen, the spores resist it and most disinfectants. During growth, the bacilli cause the production of carbonic acid, hydrogen, sulphuretted hydrogen, and carburetted hydrogen, which gases are the cause of emphysema. In the connective tissue, the bacilli increase only in places from which blood and air are excluded as much as possible, and assume in it the form of spore-bearing or homogeneous rods. On the serous membranes the bacilli become greatly elongated, and afterwards undergo division. Only towards the end of the disease, or after death, do they penetrate into the blood, where they are then found in the form of short rods, or as simple micrococci. Their vitality is arrested by putrefaction, and altogether ceases in about two months' time. If the bacilli are dried at a temperature of from 59° to 100·4° F., before putrefaction sets in, they will permanently retain their virulence. They show a considerable power of resistance to antiseptics. When fresh, they are killed by a temperature of 212° F. in 15 minutes; and when dried, by one of 248° F. in 10 minutes. Cornevin, Chamberland, and Roux obtained positive results with experiments in rendering animals immune against this disease. Besides the genuine œdema-bacilli, there are various pseudo-œdema bacilli (earth bacilli, fæcal bacilli).

Anatomical and Clinical Conditions.—The principal changes in malignant œdema consist in an œdematous, doughy and painful swelling in the neighbourhood of the infected part, which soon spreads, and is often found to crackle on palpation. In the interior, it is generally relaxed, cool, and painless; but tense, hot and painful on its periphery. The subcutaneous connective tissue, adipose tissue, and neighbouring muscles are infiltrated with yellow gelatinous material, and contain fœtid gas bubbles. The orange-coloured fluid of the œdematous swelling contains numerous characteristic bacilli of œdema and threads, which are absent in the blood during life, and are found after death only in small numbers. Besides, we find inflammation of the mucous membrane of the small intestine and œdema of the lungs. The serous fluid in the alveoli of the lungs contains great quantities of bacilli and threads. The spleen, liver and kidneys are unaffected, and it is noteworthy that the spleen is not swollen. The disease usually runs a fatal course in from 24 to 48 hours, with very severe fever. When only a few of the bacilli of œdema are inoculated, recovery with local formation of abscesses may occur.

Differential Diagnosis.—Kitt draws attention to the great similarity between malignant œdema and quarter-ill. The so-called puerperal anthrax especially, which usually appears

as a puerperal malignant œdema of the valva and vagina, is often confused with true quarter-ill. The spread of the latter is, however, restricted within enzoötic limits; but the bacilli of the former are ubiquitous and show a characteristic thread formation. In further contrast to œdema-bacilli, cultures of anthrax-bacilli in gelatine often produce no liquefaction; in the chilled blood-serum of cattle, when employed as a nutrient medium, they cause no formation of gas; in all the cultures there is no development of the phenomena of decomposition, and dextrose is not changed in alcohol. Besides, the cultures of these two varieties of bacteria can be distinguished by sero-diagnostic methods. In serous fluid, and in guinea-pigs inoculated on the peritoneum, elongated forms of the œdema-bacillus may be recognised; which, as a rule, are absent in anthrax (Leclainche and Vallée). We distinguish malignant œdema from anthrax by the fact that the bacilli of the former are not present in the blood of living animals, or in that of the fresh cadaver; are occasionally motile; have rounded ends; and that cutaneous inoculations with their cultivations in mice and guinea-pigs give negative results. See also Differential Diagnosis of Anthrax, Vol. II., p. 547.

Therapeutics.—The treatment of malignant œdema is chiefly surgical, and consists in making incisions into the emphysematous parts so as to admit air, the oxygen of which is the best agent for destroying the bacilli of œdema; in obtaining drainage; and in freely disinfecting the subcutaneous tissue. According to Leclainche and Morel, a serum of immunity for malignant œdema can be produced (intravenous injection of increasing doses of cultures, raised on Martin's bouillon).

Kitt surmises that the bacilli of malignant œdema may be the cause of many diseases which are at present classed under the respective headings of septicæmia, bovine erysipelas, and the often inexplicable œdema of the lungs of cattle. It is certain that some cases of septic puerperal fever in cattle and sheep, and probably many so-called phlegmonous swellings of surgery; as, for example, the "progressive cellular tissue inflammation" of Haubner, certain forms of the "Einschuss" of the horse, and the so-called "fire" of sheep, are simply malignant œdema. According to Haubner, the lesions of the limbs observed after the bites of sheep-dogs may, apparently, be referred to a corresponding origin; also those which develop at the seat of phlebotomy or of rowelling. The disorder appears more especially in the autumn, when the dry stable-feeding is added to the pasturage. The symptoms arise suddenly, and consist of a painful, œdematous swelling of the skin and subcutaneous tissue, usually of the inner aspect of the hind leg—seldom of the fore-

limb—of the neck, and in the larynx, as a result of which the animals limp. There is also heavy general sickness, accompanied by fever. The swelling rapidly spreads from the leg to the abdomen and chest, the skin becomes bluish-red in colour, and afterwards passes to a violet and a leaden hue, finally, it crackles on pressure with the hand, becomes cold, insensible and completely necrotic. Dullness of the senses and stupefaction increase, the animals at last lie motionless on the ground, and die, as a rule, after an average period of sickness of from 18–24 hours, rarely after 2 or 3 days. When the disease is located in the neck, angina-like troubles are noticed ; diarrhœa and the passage of bloody urine sometimes occur also. On dissection, there is found a sanguineous infiltration of the connective tissue, with development of gas, rapid decomposition of the dead body, hæmorrhages in various organs, the blood is of a dirty brown tint, and will not coagulate. There is no enlargement of the spleen.

STRANGLES.

Strangles in horses—Pseudo-strangles of dogs.

Etiology.—Strangles is an acute infective equine disease which consists essentially of an infectious catarrh of the mucous membrane of the upper air passages, especially of the nasal cavity, with suppuration of their lymph glands. According to Schütz, Jensen, and Sand, the infective agent is a chain-forming micrococcus (streptococcus equi), which can be easily found in cover-glass preparations of the pus of the lymph glands, after having been stained with aniline dyes, such as gentian violet. It can also be stained with Gram's solution. Rabe states that the mother-cells of the streptococci of strangles are ovoid bacilli, which, when connected together, sometimes assume a square form, and possess polymorphous properties. These bacilli may take the form of threads, or slender rods which remind us of the bacilli of glanders ; or they may be oval or even circular, and may sometimes form tetrads. In addition to the cocci of strangles, we sometimes find in the pus of this disease the common pus-generating bacteria (mixed infection). In broth, the streptococci of this disease form a flocky mass at the bottom of the vessel ; and on blood-serum, at 98.6° F., they form glassy, greyish droplets, and later on, a dry, iridescent crust. They have a very pathogenic effect on white and grey domestic mice, and produce suppuration in the site of the inoculation, and metastatic processes by means of the lymphatics and blood vessels (pus-forming coccus). When pure cultivations of the streptococci

of strangles are inoculated in horses, they produce abscesses in the site of the inoculation ; and strangles, when they are introduced into the nasal cavity. Cattle, sheep, pigs, dogs and birds are not susceptible to inoculation. Conclusive tests as to the virulence of the cocci of strangles are wanting, but it would not appear to be very great, for the cultures soon lose their power, and the dried pus of strangles ceases to be infectious to mice (Kitt). On the other hand, dried peelings of the skin were shown to be virulent on inoculation. (Nocard.)

Pathogenesis.—Strangles is contagious, and, perhaps, also miasmatically contagious. Schütz states that the cocci of strangles form arthro-spores, namely, permanent cells, which probably exist, and which may also grow under specially favourable circumstances, outside the animal body. Successful experiments in the transmission of strangles have been made many years ago, as for instance, by Lafosse, in 1790 ; Viborg, in 1802 ; Erdelyi, in 1813 ; and by Reynal, Toggia, and others. The contagium is found principally in the nasal mucus, and becomes disseminated in the air. Even in the last century, Erasmus Darwin assumed that a miasma which is distributed in the air is the exciting cause of strangles.

Strangles is peculiar to horses, asses, and their hybrids. It appears sporadically, enzoötically, and epizoötically, and most frequently attacks horses of from 2 to 5 years of age. Older horses, even up to 25 years of age, as was shown by Jensen, Sand, and others, may also suffer ; and also foals during the first few weeks or months after birth. According to the observations of Nocard, Wiart, Sourdel, Choisy, and Frederik, strangles may be transmitted *in utero*. In such cases, the streptococci pass from the mother to the fœtus, the internal organs of which will then contain numerous suppurating foci with the characteristic streptococcus of strangles. Similar cases have been observed in former times. According to Jonsson, strangles is unknown in Iceland, which fact can be easily explained by the contagious character of the disease. One attack confers immunity for a couple of years at least, and frequently for the remainder of the horse's life.

Youth is the chief predisposing cause ; for susceptibility to the disease is inversely proportionate to the age of the animal ; although, as before mentioned, it is never absolutely effaced. As other predisposing causes, we may mention : weakening of the vital powers by chill, catarrhal affections of the mucous

membrane, bad climatic conditions, especially change of weather in spring and autumn, pampering, continued confinement in the stable, over-exertion, transport over long distances, and an irregular system of stable management. The disease spreads epizoötically, particularly when large numbers of horses are kept together, as in cavalry depôts, in the army, in the stables of horse-dealers, and in breeding establishments.

The contagium is absorbed chiefly through the respiratory mucous membrane. The high initial internal temperature, which is manifested even prior to the swelling of the glands, and the experimental observations of Schütz, Jensen, and Sand, prove that the infective matter may be taken into the blood directly from the mucous membrane. In its further progress, the contagium keeps to the direction of the lymph passages and enters by preference into the lymph glands, in which it excites extensive suppuration. As was shown by Schütz, the cocci of strangles can also pass through the walls of the capillaries without the help of the blood or of the lymph, and can penetrate between the tissue cells of the affected organs. It is probable that the contagium may enter the body also from the intestinal mucous membrane. This supposition is strengthened by the fact of the occurrence of abscesses in the mesenteric glands, with a normal condition of the nasal mucous membrane and the simultaneous absence of abscesses in the submaxillary gland, by the existence of abscesses in the submucosa of the intestinal mucous membrane, and by changes in Peyer's patches and the solitary glands, which can sometimes be demonstrated. The same remark applies to the lungs, the lymph glands of which, namely, the bronchial glands, are often found to be in a suppurating condition. Infection sometimes takes place, also, in some cases, from wounds of the skin, and through the udder (as in case of suckling foals suffering from strangles).

The period of incubation appears to vary from 4 to 8 days. By the simultaneous influence of cold, the time is rendered much shorter, and reaches then a minimum of one day (predisposing catarrh).

History.—The earlier views on the nature of strangles are now only of historical value. This disease was regarded as due to chill or development, or was connected with a supposed lymphatic equine idiosyncrasy. Dentition and a peculiar state of the weather were also regarded as causes of it. Others consider strangles to be identical with scrofula or with human measles. In more recent times, Trasbot,

as Sacco had previously assumed, declared that strangles were true horse-pox. Trasbot, as Viborg and Toggia had done in their time, proposed that all horses should be inoculated with the lymph of horse-pox as a prophylactic measure against strangles. This hypothesis, which is in itself quite improbable, has been refuted experimentally by Delamotte, who demonstrated that the inoculation of horse-pox does not protect horses against strangles. Furthermore, the fact of the existence of different forms of strangles was advanced; as for instance, strangles of foals; benign, suspicious, malignant, slow, metastatic, occult, gangrenous, putrid, asthenic, compound, and vesicular strangles; strangles caused by infectious matter which had to be eliminated from the body; strangles originating from glanders; etc. The early crude appellations of the disorder were: " throat disease "; " laryngeal disease " or " swollen larynx " ; adenitis or morbus glandulosus equorum.

Symptoms of ordinary, mild Strangles.—An attack of strangles begins by a rise of internal temperature varying from 104° to 105·8° F. on an average. During the following days, the temperature falls one or two degrees, and does not again rise until suppuration takes place in the lymph glands. It decreases simultaneously with the outward discharge of pus. At the commencement of the disease, the pulse usually remains more or less normal in spite of the high temperature, and only later on, its rate increases to 50 or 60 beats a minute. A higher frequency is met with only in animals of very weak constitution and when complications arise.

The first local symptom consists in catarrh of the nasal mucous membrane, which generally becomes diffusely congested; although, at times, the redness may be confined to certain spots. The discharge is at first serous and viscid; but after about three days it becomes mucilaginous and finally purulent, and then its colour varies from a dirty-white to a yellowish green. This purulent catarrh is usually found in both nasal cavities, but may, in exceptional cases, be more or less unilateral. In young animals, it is generally very abundant, but in aged horses sometimes very slight.

In the large majority of cases, swelling of the submaxillary glands appears concurrently with the purulent nasal catarrh. This swelling is hot and painful to the touch. The spreading of the inflammation to the connective tissue which surrounds the glands, and the stasis of the lymph in the efferent lymph vessels, often cause the development, from the submaxillary glands, of very extensive swellings that may occupy the entire inter-maxillary space, and may spread even to the exterior side of the

posterior maxilla. The course of the swelling of the submaxillary glands may vary. In by far the greater number of cases, abscesses form; but we may sometimes have dispersion and absorption with gradual resolution or induration. The formation of an abscess becomes known by the appearance, in the swelling, of soft spots, which at first are indolent, but later on are distinctly fluctuating. The skin over these spots is tense, purple in colour, and covered with a lymph-like viscid exudate. The hair over these spots falls out; the skin covering them becomes thin, discoloured and necrotic; and the abscess "points," spontaneously bursts, and discharges from one or more openings a thick, yellowish-white, creamy pus, which is sometimes mixed with necrotic shreds of tissue. The abscess cavity heals by granulation. As soon as the abscess bursts, which it will generally do in from 8 to 12 days, if it has not been previously opened, the swelling will rapidly disappear from the part, and the internal temperature will fall as quickly as it rose. In exceptional cases, strangles may present only catarrhal symptoms without suppuration of the lymph glands. Jensen states that it may first assume the form of pharyngitis, purulent pneumonia, and pleuritis, without any well-marked morbid affection of the lymph glands.

Among accompanying symptoms we may mention: loss of appetite, which is partly due to the general feverish affection, and partly to the swelling of the intermaxillary space, depression, occasionally, excessive weakness at the beginning of the attack, œdematous swellings, especially of the hind-legs, and often great emaciation, if the disease has lasted a considerable time. In spite of the very high temperature, the urine generally remains alkaline; although it frequently contains a considerable quantity of albumen. During convalescence, we may sometimes observe a temporary polyuria, as in other acute infective diseases.

Finally, strangles is accompanied by a cutaneous exanthema which usually takes the form of a rash the size of a pea up to that of half-a-crown, and spreads over the entire body; or an eruption of nodules, vesicles, and even pustules, chiefly on the lateral surface of the neck, shoulders and sides of the chest. These exanthemata are characterised by their sudden appearance, and often by their equally rapid disappearance. An eruption of vesicles may break out on the nasal mucous membrane, and, in exceptional cases, may spread to the skin of the alæ nasi and upper lip. The contents of the vesicles are at first limpid, and become purulent later on. The

vesicles soon burst, and leave shallow ulcers, which become covered with a thin scab and heal without leaving a scar. These vesicular processes on the nasal mucous membrane give rise to the idea that they are characteristic of a peculiar kind of strangles, for which, some writers have evidently mistaken cases of stomatitis pustulosa contagiosa, and frequently those of inflammation of the lymphatics. Elevations on the nasal mucous membrane resembling wheals are rare. They take the form of circumscribed, dark-red, raised, sero-hæmorrhagic infiltrations of the mucous membranes, varying in size from a lentil up to a pea, and are often surrounded by a red-coloured edge. Rabe states that the streptococci of strangles can produce ulcers on the nasal mucous membrane in consequence of croupy and diphtheritic infiltration of the mucous membrane (see remarks on the Differential Diagnosis of Glanders, Vol. II., p. 480).

Complications of Strangles.—1. One of the most frequent complications is the spreading of the infectious catarrh to the *pharyngeal mucous membrane*, with the consequent production of *pharyngitis*, which, in a few cases, may be the first symptom of strangles. Pharyngitis, thus transferred, is well marked. The patient suffers from difficulty in swallowing, salivation, regurgitation, and swelling and suppuration of the retro-pharyngeal lymph glands (the so-called upper cervical glands or subparotideal glands). The affection of these glands manifests itself outwardly by a diffuse and often very extensive swelling in the lower parotid region, above the larnyx ; and the lymph glands near the temporo-maxillary articulation directly under the ear, and the lymph glands lying between the lobules of the parotid gland, may also participate in the inflammation. These glandular swellings usually terminate in suppuration and discharge of pus, partly outwards and partly inwards in the direction of the pharynx and larynx. In other cases the abscess undergoes caseation and becomes partly absorbed, or becomes fluid with subsequent septicæmia or pyæmia. The abscesses may attain an extraordinary size by several small ones becoming confluent, and the connecting tissue surrounding the pharynx is often densely infiltrated with suppurating foci. Smaller abscesses occur under the pharyngeal mucous membrane and in the muscular apparatus of the pharynx. A phlegmonous abscess in the subcutis may be developed from the retro-pharyngeal lymph glands, and may give rise to extensive swellings of the skin of the head and neck. Under the most favour-

able circumstances, the pus in the suppurating lymph glands works outwards, either directly or after burrowing a way for itself; or it may burst inwards into the pharyngeal cavity by reason of the mucous membrane having become necrotic on account of pressure exercised by the abscess, in which case the production of gangrenous pneumonia is a frequent sequence. The abscess may open inwards as well as outwards, and thus cause a pharyngeal fistula. Fistulæ may form in a similar way from the submaxillary glands, and may in a few cases open into the oral cavity.

2. The infectious inflammation may spread from the nasal cavities to the *mucous membrane of the larynx, trachea, bronchi, accessory cavities of the nose, oral cavity, eyes, and guttural pouches.* A superficial catarrhal inflammation, the irritation from which sets up cough, develops at first in the larynx, and, sometimes, on spreading downwards, becomes phlegmonous, and may thus lead to severe dyspnœa. In the course of this *laryngitis* we may have the formation of submucous abscesses, inflammation and paralysis of the muscles of the larynx, and roaring, or "whistling." The catarrh of the bronchi may lead to swelling and suppuration of the bronchial glands. Masses of fodder are, in a few cases, found wedged in the guttural pouches in consequence of necrosis of its walls, and may penetrate even into the subcutaneous tissue along the throat around the trachea. In strangles the guttural pouches are, however, rarely implicated.

3. Inflammation of the *superficial lymph vessels of the skin of the head,* with the formation of a large number of minute abscesses, is chiefly met with on the cheeks, lips, alæ nasi, and sometimes over the entire head as far as the ears, even down to the throat. The numerous lymph vessels of the implicated skin are then inflamed, and the connective tissue which surrounds them becomes infiltrated with pus. This perilymphatic suppuration produces in the course of the lymph vessels, numerous minute abscesses which spring from rosary-like nodules that have nothing to do with the suppuration of the lymph glands. This inflammation of the lymph vessels may be followed by diffuse, phlegmonous swelling of the lips and cheeks, in which case the mucous membranes of these parts become tense and frequently as hard as a board. There may also be simultaneous suppuration of the mucous glands which, on the eversion of the tensely swollen lips, will appear as plugs of pus, and will give rise to minute ulcers. The formation of an eczematous eruption on the head is also occasionally observed.

vesicles soon burst, and leave shallow ulcers, which become covered with a thin scab and heal without leaving a scar. These vesicular processes on the nasal mucous membrane give rise to the idea that they are characteristic of a peculiar kind of strangles, for which, some writers have evidently mistaken cases of stomatitis pustulosa contagiosa, and frequently those of inflammation of the lymphatics. Elevations on the nasal mucous membrane resembling wheals are rare. They take the form of circumscribed, dark-red, raised

groove, which swelling may sometimes burst on both sides and may cause roaring); omo-hyoideus and sterno-hyoideus muscles, lower cervical glands (with formation in front of the entrance of the trachea into the thoracic cavity, of a tumour which may cause compression of the trachea, and suffocation or permanent stenosis of the trachea); axillary glands (producing lameness), subcutaneous tissue, especially on the lateral thoracic walls, front of the chest, lower ribs, hypogastrium, flanks, scrotum, udder, neck, withers, inner surface of the thighs, etc. These subcutaneous abscesses are accompanied by extensive swelling of the skin of the affected parts, the condition of which is frequently mistaken for mere œdematous swellings of the skin. Besides the swelling and suppuration of the bronchial glands, we find in the thoracic cavity metastatic mediastinal abscesses which vary in size from a pea to a man's head, and which produce by their rupture a purulent pleuritis (empyema). Similar abscesses, which often contain as much as 2 gallons of pus, and give rise to fatal peritonitis by their perforation into the abdominal cavity, may be found in the mesentery, omentum, pelvic connective tissue, mesenteric glands, pancreas, kidneys, etc. These abscesses may lead in some cases to adhesions between the intestines. Their presence is manifested during life by periodic and often chronically recurrent slight attacks of colic, and may, in a few cases, be ascertained by exploration *vià* the rectum. Abscesses which are accompanied by swelling and suppuration of the intestinal follicles, and which are also characterised by frequently occurring remittent colic, are sometimes found under the mucous membrane of the stomach and intestines. Numerous abscesses of greater or less size and purulent infiltration may be observed in the brain (with symptoms of inflammation of the brain, chronic hydrocephalus, blindness, and local symptoms); spinal cord (paraplegia of the hind limbs, and paralysis of the rectum and bladder); joints, especially in the knee, hock, fetlock, and coronary articulation (with suppurating synovitis, swelling, and functional disturbances);

able circumstances, the pus in the suppurating lymph glands works outwards, either directly or after burrowing a way for itself; or it may burst inwards into the pharyngeal cavity by reason of the mucous membrane having become necrotic on account of pressure exercised by the abscess, in which case the production of gangrenous pneumonia is a frequent sequence. The abscess may open inwards as well as outwards, and thus cause a pharyngeal fistula. Fistulæ may form in a similar way from the submaxillary glands, and may in a few cases open into [illegible] animals which have been thus affected sometimes continue permanently lame after recovery from strangles.

5. Complications may arise in the lungs in the form of suppurating (metastatic) *pneumonia* due to foreign bodies and simple catarrhal pneumonia. Generally speaking, pneumonia caused by foreign bodies in cases of pharyngitis, is set up by the entrance of the pus of pharyngeal abscesses into the air passages. There is a metastatic pleuritis also developed in some cases. Finally, contagious equine pleuro-pneumonia may also be a complication of strangles (compare the pyæmic abscesses of the lungs in the course of this disease, in which the streptococcus of strangles may be identified [Jensen]).

6. Strangles assumes a chronic lingering course, when a *chronic catarrh*, which keeps up the nasal discharge, becomes established in the sinuses of the head, and in the guttural pouches, or in the pharyngeal cavity. The animal becomes very thin and suffers from indigestion, and frequently appears as if it was affected with glanders (the so-called suspicious strangles), in consequence of the similarity of these symptoms with those of chronic glanders.

7. Death from strangles is usually caused by the additional influence of *septicæmia* or *pyæmia*. In such cases, there is high fever and great depression, the pulse is frequent and weak, palpitation of the heart ensues, the animal becomes debilitated, often in a very rapid manner, and severe diarrhœa sometimes sets in towards the end.

8. Strangles is sometimes complicated with *petechial fever*, most frequently during the stage of convalescence.

Differential Diagnosis.—Strangles is mistaken chiefly for simple nasal catarrh which is accompanied by a purulent nasal discharge, and which is tolerably frequent, particularly in young animals. It is distinguished from strangles by the fact that it never leads to suppurating inflammation of the sub-

maxillary glands, although in a few cases it may cause swelling of these glands. To distinguish strangles from glanders, see the differential diagnosis of glanders, Vol. II., p. 480, *et seq.* In doubtful cases it is advisable to inoculate white mice, which are immune from glanders; although they are typically susceptible to the inoculation of strangles. Strangles may also be mistaken for parotitis and mucous degeneration of the turbinate bones.

Prognosis is favourable; because the mortality is small. Thus, out of 5,520 remounts and horses in the ranks of the Prussian army which suffered from strangles in the years 1888 to 1893, only 2 per cent. died. In the Bavarian army during the last two years there was a mortality of 2·5 per cent. Ringsheim states that among 2,205 horses of the Danish army which suffered from strangles, only 0·5 per cent. died during the years 1851 to 1860. This surprisingly low percentage may be explained by the fact that the ages of these horses were from 4 to 9 years. Krabbe on one occasion found among 1,789 affected horses of every age, a mortality of 1·6 per cent.; and at another time among 1,107 sick horses, one of 3·5 per cent. During 1885, in Denmark, Goldschmidt observed a mortality of 2·3 per cent. among 2,419 affected horses. During 1883, the mortality in Denmark was 2·6 per cent. among 2,381 affected horses. We may consequently assume that the average mortality of strangles is from 2 to 3 per cent. Among favourable signs we may mention a robust constitution and a normal development of the disease, and among unfavourable ones: debility from previous illness, youth, and complications, especially metastatic pyæmia, and ichorous pneumonia.

Therapeutics.—In normal cases, it is sufficient to attend to hygiene, which consists in cleanliness, assiduous ventilation of the stable, and the administration of food which can be easily digested, such as "green meat," mashes, and roots. It is useless to give febrifuges to reduce the initial high internal temperature, which will abate of itself under normal conditions in a few days. The treatment of the suppurating lymph glands is purely surgical, its essential principle being to open the abscesses with a long incision as early as possible, in fact, as soon as pus forms in them. The early discharge of the pus is quickly followed by a fall of the internal temperature, and evidently shortens the duration of the disease; consequently we may reject the old

custom of allowing the abscesses to "ripen" in a natural manner. If the swelling is indolent, we may stimulate the formation of pus by fomentations or blisters.* We should open the abscess with caution on account of the proximity of numerous blood vessels, and should divide only the skin with the knife, and then work up to the abscess with the finger. Failing to penetrate the abscess, it is often necessary to make a second attempt in another direction in the event of our not wishing to explore with a trocar and cannula.

For catarrh of the mucous membranes in strangles, practitioners generally use inhalations of steam and internal remedies, such as preparations of antimony (crude sulphide of antimony and orange), and alkalies (sulphate of soda, chloride of ammonium, bicarbonate of soda) combined with liquorice, honey, etc. If the catarrh proves obstinate, we may give in each feed a teaspoonful of the officinal artificial Karlsbad salt, or an electuary of sulphate of soda 8½ ozs., crude sulphide of antimony ⅞ oz., with liquorice powder and althæa powder, twice a day. In accordance with the nature of the case, the most varied combinations of individual remedies are possible. The so-called "strangles-powder" which enjoys public favour, frequently does contain similar medicaments; but consists, in most cases, merely of decomposed vegetable powders which have become useless; and which, therefore, more often cause injury than improvement. On this account people should be cautioned against them. Since Träger did this more than fifty years ago, every text-book of veterinary pathology has contained the warning, although manifestly without result.

Complications must be treated according to their nature. Tracheotomy has sometimes to be performed to relieve excessive dyspnœa. For cases in which pharyngitis supervenes, Kagel recommends the introduction of a strong indiarubber tube of about 2 ft. in length into the nostrils as far as the upper end of the nasal cavity, with subsequent injection of about 5 ozs. (50 c.c.) of a disinfecting solution. From our own experience we can speak favourably of this local treatment. Dieckerhoff recommended intravenous injections of argentum colloidale

* I have had most excellent results, by strongly blistering the swelling, as soon as it appears, with biniodide of mercury ointment (1 to 4 of lard). I am inclined to think that, in these cases, the good effect of the blister is due to the high antiseptic action of the biniodide of mercury, which, according to Cadéac is 34 times greater than that of corrosive sublimate.—TR.

(6 grains in a 1 per cent. solution) when the course was prolonged, in cases of phlegmonous swellings and in septic fever. This remedy has not, however, proved satisfactory; for of the foals affected with strangles which were treated with it, more died than of those which were not so treated (Baumgart, etc.). In the Prussian army, also, the remedy was often found as ineffective as the yeast treatment recommended by Ludewig when tried in the remount-depôts. Ichthargan has lately been advocated by Bass.

Finally, for prophylaxis, the disinfection of the tainted stables, and the separation of healthy from affected animals are necessary. How far serum-therapy (anti-streptococcus serum, strangles-serum, gurmin) may be relied on in the way of prophylaxis and cure still remains to be demonstrated (Delvos, Jess, and Piorkowski). Schnürer has found neither in the serum of horses spontaneously affected, nor in the active serum of immunised animals, any protective matter against infection with the streptococci of strangles.

Inoculation.—In various directions the question of the artificial production of strangles in healthy animals by inoculation, with a view to immunity, has been brought up for discussion. The experiments made by Jensen and Sand with intravenous injections of the coccus of strangles, produced no general infection, but only violent cellulitis, which, however, conferred immunity from subsequent infection through the nasal mucous membrane. We have to wait for further experiments with this method before giving a decisive opinion on it. It is certain that in Germany, Peterson's very peculiar method of producing infection will not find many disciples. He exposed all the foals of a breeding establishment during autumn to severe cold, by driving them into a pond and keeping them there for half an hour, and then took them to a hill, where they were exposed on all sides to cold winds. After that, he gave them very cold water to drink. All these animals became affected with strangles, but recovered completely in 3 weeks.

Strangles of Dogs.—Puppies may suffer from a purulent follicular inflammation of the lips with purulent lymphangitis and purulent lymphadenitis of the head, which may lead to pyæmia, and which consequently possesses a certain similarity to strangles. As it is an original infective disease which is independent of distemper and pyæmia, we may give it the name of "canine strangles." In cattle, too, isolated cases of a disease resembling strangles are known to occur. According to Peschke, in some stalls the calves sickened with multiple-abscess formation in the head. Nagy saw in two cows suppuration of the laryngeal lymphatic glands with purulent nasal discharge and general febrile disturbance.

PETECHIAL FEVER.

(*Horse typhus, Morbus maculosus. Purpura hæmorrhagica.*)

Petechial fever in horses—Typhus of man—Petechial fever in cattle.

Former Views on Petechial Fever.—Every infectious disease which was accompanied with decomposition of the blood and severe general cerebral symptoms was formerly known as typhus or putrid fever. These were more especially splenic fever, septicæmia, influenza, epidemic pleuro-pneumonia, acute glanders, cerebral meningitis and typhoid diseases. People spoke of a typhoid stage of this or that disease, when during the course of the symptoms, notable functional disturbances of the sensorium appeared. In this sense petechial fever was originally reckoned as typhus, and was specially named "equine typhus," or petechial typhus, for the purpose of more exact discrimination from other typhoid diseases, which names have been retained to the present day. In addition to these, the term putrid fever came into general use, and was employed by Hertwig in 1836, and afterwards by Spinola and others. Hering called it "petechial fever" (a term which we have retained), after its most important symptom, viz., that of hæmorrhages in the mucous membranes. He, however, laid particular stress on the fact that petechiæ on the mucous membranes also occur, as a mere symptom, in diseases, such as strangles, which have no connection with petechial fever.

Accordingly, the term typhus has, as will readily be understood, led to the supposition of an identity of equine typhus with the abdominal typhus and the spotted typhus of man (concerning the error of this view, see *infra*). In England, petechial fever was regarded by some as scarlatina; by others, as morbus maculosis of man (purpura hæmorrhagica). The name of morbus maculosus was commonly applied to it in Germany, as for instance by Eberhard. In France and Italy petechial fever is regarded as acute anasarca; and was traced, not to decomposition of the blood, but to great relaxation and temporary paralysis of the capillaries by the effect of vaso-dilatory toxins, and consequent effusion of serum and blood. According to Lignières (1895) petechial fever is to be regarded as an infection produced by the streptococcus pyogenes, (Lafosse and

others had previously stated that it was either septicæmia or anthrax).

Owing to the example of Röll, petechial fever was supposed for a long time in Germany to be an anthracoid disease. Hering, Haubner and others have, however, prominently emphasised the fact that this theory is erroneous; because it is neither infectious, nor can it be transferred by inoculation to horses or to other animals. Besides, the bacilli of anthrax have never been found in the blood of horses suffering from it, and some of its symptoms are incompatible with the supposition of anthrax. Arloing failed to transmit it, even by transfusions of blood.

Dieckerhoff considered that petechial fever was similar to morbus maculosus Werlhofii of man, and proposed that the name, "morbus maculosus," which was already in use, should be retained. We prefer the designation of petechial fever, and leave undecided the question of its relation to morbus maculosus, the causes and nature of which are not yet clearly known. We agree, however, with Dieckerhoff that the names of "horse typhus" and "petechial typhus" should not be employed; for petechial fever has nothing in common with the typhus of man.

Typhus in Man.—Three varieties of typhus are distinguished in man: abdominal typhus or ileotyphus, exanthematous or spotted typhus (which is also called petechial typhus), and relapsing typhus (typhus or febris recurrens).

1. Abdominal typhus, the usual form of typhus, is produced by a specific bacillus, and is a contagious miasmatic disease, which essentially consists of a necrotic infiltration of the follicular lymph-apparatus of the small intestine, especially of the ileum in the neighbourhood of Bauhin's valve, with pronounced enlargement of the spleen, and also catarrhal affections of other mucous membranes. It begins with rigors and marked psychic disturbances, followed by a typical fever, persistent diarrhœa, bronchitis and grave nervous conditions (delirium, twitching of the tendons, etc.), accompanying it. A few isolated reddish spots (3–6 in number) appear on the skin of the chest and abdomen. Several complications may also arise, such as: intestinal hæmorrhage, intestinal perforation, angina, laryngeal ulceration, parotitis, grave pulmonary inflammations, nephritis, affections of the brain, embolism, paralysis, etc., the proportion of fatal cases at present reaches but about 7 per cent. The experimental administration of the excrements of persons affected with abdominal typhus to animals with their food has never yielded any result. Even the intravenous injection of virulent cultures of typhoid bacilli is without effect on the dog; the animals remain perfectly healthy, and develop neither fever nor diarrhœa. Moreover, according to the experimental researches of Baumgarten, the human typhoid bacilli were not inoculable on the rabbit, or the guinea-pig or mouse. Accordingly

they cannot be regarded as pathogenic in relation to these animals. The cases reported in literature of accidental communication of abdominal typhus to animals are mostly based on a confusion with splenic fever (*e.g.*, the cases reported by Ammon), or with mycotic inflammation of the intestine and with other septic diseases; possibly, also with poisoning. Of veterinary authors, Semmer alone expresses himself in favour of the occurrence of human typhus in domestic animals.

2. The petechial typhus of man is an infectious disease which is communicable in the highest degree, and prevails in some localities as an endemic affection (so-called famine-typhus or war-typhus). On *post-mortem* examination, there are found no characteristic alterations, only very general ones; the characteristic intestinal lesions of abdominal typhus being conspicuously absent. On the other hand, the course of the disease is very typical; and there appears on the skin a roseolar exanthema, from which there are afterwards developed petechiæ, *i.e.*, true hæmorrhages. The crisis and convalescence are very soon passed. The course is rather favourable (mortality about 10 per cent.).

3. Relapsing typhus is produced by the corresponding spirillum (spirochæte Obermeyeri), which is found in the blood. It also runs its course without any special lesions of the organs, its chief symptom being a sudden attack of high fever. The course is very favourable (mortality, 2 per cent.).

Etiology and Pathogenesis.—Petechial fever is an acute infective disease, the cause of which is unknown. Specially liable to objection is the view that it is an infection due to the streptococcus pyogenes, which is the usual cause of suppuration, phlegmonous inflammation, and septicæmia. It is characterised by numerous hæmorrhages in the skin, subcutis, mucous membranes, submucosa, and internal organs, with subsequent inflammatory œdematous swellings of the skin, mucous membranes, subcutis, and submucosa. Although the disease may appear primarily, it is usually a sequela to previous infective diseases, such as strangles, pharyngitis, contagious equine pleuro-pneumonia, and influenza, and, chiefly, during convalescence after these diseases. Chronic catarrh of the lungs, chronic inflammation of the maxillary sinuses, necrosis of bone, subcutaneous abscesses, fistulæ of the withers, castration wounds, intestinal catarrh with suppuration of the lymph follicles, abscesses in the lungs, kidneys, and spleen, have been observed to be the starting-points of this disease. It sometimes follows a slight injury to the skin (scratches or punctures). We have, moreover, observed that petechial fever has developed from wounds in process of healing, and which were daily undergoing antiseptic treatment. One case has been reported, in which the petechial fever had developed from a wound already healed (Sobornow).

Dieckerhoff recognises in this disease an auto-intoxication of the body, from certain primary foci—such as: suppurating lymph-glands, abscesses, suppurative catarrhs—with a chemical poison, which has formed in the foci concerned under the agency of micrococci, and by causing special irritation affect the walls of the vessels so as to lead to hæmorrhage. The pathogenesis of the disease has been similarly regarded in France (vaso-dilatory toxins, according to Cadéac). The view of a chemical poison circulating in the blood can be supported by the frequently apyretic course of the disease, the often very rapid simultaneous appearance of swellings in different localities; as well as by the facts that the disease is neither infectious nor inoculable, and the schizomycetes may not, with certainty, be present. Against the theory of a primary focus the fact must be advanced that, in several cases, a preceding primary lesion was not recognisable, the disease having arisen in perfectly healthy animals, in which the assumption of a primary focus would have been wholly arbitrary. On the other hand, we must hold that in the *post-mortem* examination of horses which have died of petechial fever, especially after prolonged and severe illness, it may not be quite possible always to determine which is a primary focus and which a secondary. Conversely, the generally rare occurrence of petechial fever in comparison with the frequency of local suppurating foci in the body (suppurating wounds, abscesses, empyemata, strangles, chronic suppurative mucous catarrh), is remarkable. In favour of the idea of a schizomycete infection are also: the similarity of the disease to malignant œdema, the occurrence of the bacillus hæmorrhagicus in the purpura hæmorrhagica of man, the occasional appearance of local groupings of cases (Zschokke has observed an apparent epidemic of the disease in cavalry remount-depôts, and the outbreak of numerous cases in the same stable).

A disease identical with petechial fever of horses appears to occur in cattle and goats, especially in cases of inflammation of the uterus, mastitis, etc. We have several times noticed in dogs, hæmorrhagic affections of the character of human morbus maculosus; but no ailment which corresponded to the petechial fever of horses.

Bacteriology.—Kolb recently found, 3 or 4 hours after death, in people who had suffered from morbus maculosus (purpura hæmorrhagica), a bacillus to which he gave the name of "bacillus hæmorrhagicus." This bacillus forms oval, thick, non-motile rods with rounded ends, in

the form of diplo bacilli, attached to one another, and sometimes forming pseudo-threads. They cannot be stained with Gram's solution. Pigeons and guinea-pigs were found to be immune; but dogs, rabbits, and mice became affected with a hæmorrhagic ailment. The bacilli were seen in great numbers in the blood of the cadaver. Letzerich states that he has discovered the specific bacillus of morbus maculosus. How far these statements will obtain confirmation, and whether the bacillus hæmorrhagicus is also present in the petechial fever of horses, still remains to be determined.

According to Lignières, petechial fever is produced by a streptococcus which is identical with the streptococcus pyogenes. Cadéac states that it may come from any bacterium which produces vaso-dilatory toxins. Mouilleron and Rossignol hold that there are, in addition to the streptococcus, numerous other bacteria present; they reduced the mortality to 19 per cent. of 31 horses affected, by the use of anti-streptococcus serum (5 drms. to 3 oz. 7 drms. injected daily). Frasey observed on injecting two horses with hypervirulent cultures of streptococcus pyogenes, that they sickened with symptoms of petechial fever. On the ground of his experiences in South Africa, Theiler has come to the conclusion that influenza has a causal connection with petechial fever. Bernabei and Boatini have observed the simultaneous occurrence of petechial fever and of splenic fever in Italian horses.

Anatomical Conditions.—The anatomical changes consist in hæmorrhages in the skin, subcutis, mucosa and submucosa of the nasal cavity, larynx, trachea, bronchi, conjunctiva, pharynx, oral cavity, stomach, intestine, bladder and vagina, lungs, spleen, kidneys, and muscles of the heart and skeleton. Zschokke assumes as the cause of these hæmorrhages, a mycotic endarteritis with the formation of thrombi and subsequent infarcts. These changes, as in poisoning by phosphorus, may be due to fatty degeneration, and brittleness of the arterial walls in consequence of injury caused by the virus. The smallest hæmorrhages are found in the nasal mucous membrane, where they sometimes vary in size from a millet seed to a lentil. The largest are in the lungs, where they may attain the size of a man's fist. As a consequence of the hæmorrhage, we find inflammatory sero-gelatinous swellings in the subcutis, submucosa, and inter-muscular tissue, which may lead even to sloughing of the affected parts, especially of the skin of the flexor surfaces of the joints, intestinal mucous membrane, and less frequently of the mucous membrane of the nose and pharynx. Hæmorrhages into the cavities of the body and even death may occur from laceration of hæmorrhagically infiltrated parts, or a parenchymatous inflammation may become developed, as for instance, in the lungs. Owing to gangrene of the inflamed tissues, pyæmia, septicæmia, or perforative intestinal peri-

tonitis may set in. The individual organs show the following changes :—

1. The *skin*, especially on the most depending parts of the body, as on the legs, muzzle, brisket and abdomen, becomes locally or diffusely swollen, and infiltrated with hæmorrhages, which vary in size from a pea up to half-a-crown, and which give the skin a spotted appearance after it has been removed from the body, *post-mortem*. The skin over the swollen parts frequently shows lacerations and sores which may penetrate into the muscles. The subcutaneous cellular tissue appears on section to be infiltrated with serous fluid and blood, and is also considerably swollen.

2. The *mucous membrane of the nasal cavity* is covered with irregularly shaped blood spots which vary in size from a lentil up to a hazel nut, and which frequently become confluent and thus form large sanguineous blotches. We sometimes find the entire nasal mucous membrane infiltrated to such an extent with blood, that the lumen of the nasal cavities is considerably narrowed. The infiltration is usually greatest in the mucous membrane of the turbinate bones. The nasal mucous membrane may show ulcers which may penetrate so deeply as to produce necrosis of the cartilages and the formation of holes in the septum nasi. Similar infiltrations and hæmorrhages are found in the mucous membrane of the nasal cavity and larynx, with consequent œdema of the glottis. The epiglottis and the guttural pouches are the seat of a sanguineous infiltration, and ulcers appear in the pharynx as well as in the larynx. The retro-pharyngeal lymph glands frequently become swollen and suppurate. Petechiæ are not very numerous in the oral cavity in which they are chiefly found on the gums, tongue, and inner surface of the swollen lips, and may run into ulcers. The conjunctiva and the vaginal mucous membrane sometimes become swollen and covered with petechiæ. The mucous membrane of the stomach shows red spots. Hæmorrhages occur under, as well as in, the mucous membrane, under the serosa and in the muscular layers, in which they are generally so finely divided, that the muscular tissue, on section, has a greyish-blue colour. In the intestinal canal we find masses of exudate which vary in size from a lentil to the palm of one's hand, and which have been thrown off by the mucous membrane, submucosa and subserosa. The muscular coat of the intestine is infiltrated with minute hæmorrhages. The mucous membrane is more or less swollen and is frequently the seat of ulcers, which vary

in size from a quarter of an inch to four inches in diameter. These ulcers, which have a tinder-like base, sometimes granulate and form scars. At other times, they perforate the walls of the intestine and set up peritonitis. The contents of the intestine are sometimes mixed with blood. The changes are best marked in the small intestine; although they are also found in the cæcum, colon and even in the rectum, in which parts the hæmorrhages often take the form of streaks.

3. *The muscles of the skeleton*, such as those of the pelvis, thighs, abdomen, chest, tongue, the masticatory muscles, etc., frequently show great numbers of dark-brown or copper-coloured localised hæmorrhagic infiltrations of various forms and sizes. The muscular fibres often become granulated and undergo fatty degeneration, so that the affected muscle becomes pale and of a clay colour. The interspersed hæmorrhages may give a marbled appearance to the muscles. The intermuscular connective tissue is frequently gelatinously and hæmorrhagically infiltrated. Portions of muscular tissue sometimes become gangrenous and are cast off. The muscular tissue of the heart becomes similarly affected; it may contain numerous subepicardial, subendocardial, and interstitial hæmorrhages; and may assume a similar clay or greyish-red colour. There may also be hæmorrhages under the periosteum. A necrotic articular inflammation has occasionally been observed, especially in the fetlock and cubital joints (polyarthritis); erosion of the tendons has also been noticed.

4. The *lungs* are infiltrated with miliary, subpleural, and parenchymatous blood foci, which may produce a diffuse hæmorrhage of the lungs. We also observe extensive croupous, and multiple necrotic, purulent, or ichorous pneumonias (the last of which are due to foreign bodies), hypostasis of the lungs, and œdema of the lungs.

5. We sometimes find in the *spleen* and *kidneys*, large nodular hæmorrhages, the rupture of which may cause hæmorrhage into the abdominal cavity. There is no distinctive swelling of the spleen. The cavities of the body often contain moderate quantities of bloody transudate, and the abdominal cavity sometimes contains a large amount of fluid blood.

6. The *blood* shows no essential changes, as long as the disease progresses without complications. No bacteria have as yet been found during life; although the bacilli of malignant œdema can be demonstrated in it soon after death, as well as in horses which have died from suffocation.

7. Besides the above-mentioned phenomena, we find secondary changes resembling those of *septicæmia* or *pyæmia*, and of *suffocation;* and at times, the residual changes of the primary affection (strangles for instance) which preceded the petechial fever.

Symptoms.—The first symptom in horses is usually the appearance of numerous dark red *petechiæ, i.e.*, blood foci, on the more or less congested nasal mucous membrane. They are sometimes as small as a flea-bite; but are generally from one-quarter to three-quarters of an inch in diameter, and frequently run together so as to form large blotches or streaks. Owing to their presence, the nasal mucous membrane assumes a peculiar dotted or speckled appearance, and may become purple throughout its entire extent, if the petechiæ be very numerous. It is at the same time more or less swollen, and exudes serous fluid. In very severe cases the mucous membrane becomes gangrenous, and covered with ulcers; the nasal discharge is frequently sanious and discoloured; and the expired air has a fœtid odour. In such cases, respiration becomes difficult, and the disease may take a rapidly fatal course with severe aggravation of the general symptoms. The eruption of the petechiæ may continue during the entire course of the disease.

Simultaneously with, or a few days after, the appearance of petechiæ on the nasal mucous membrane, we find *cutaneous swellings* which are often the first symptom observed, on account of their being more readily seen than the blood spots. These swellings take the form of wheals, and are from three-quarters to one and a half inches in diameter. They may be distributed over the whole body, but are generally found on the most dependent parts, such as the limbs, lower portion of the face, abdomen, sheath, and lower surface of the chest. These swellings at first vary in size from an apple to a man's fist, and resemble carbuncles. Later on they become confluent or spread upwards, so that we have sharply defined diffuse swellings, which greatly disfigure the attacked parts. The extremities increase in circumference; large tumours form on the lower surface of the chest and abdomen, and the lower part of the face becomes like that of a hippopotamus. The swellings are hard and painful to the touch, and when they occur on the head they are sometimes as hard as a board. The hair on them often falls out. The nose-band of the halter frequently makes an indentation in the swelling on the nose. The rapid and

simultaneous appearance of swellings on several parts of the body is characteristic of this disease. When the swelling is severe, lacerations of the affected skin of the flexor surfaces of the articulations are very liable to occur. Cutaneous gangrene may supervene, with consequent sloughing of pieces of skin, and the formation in their place of indolent ulcers. The smooth, dense skin, first of all exudes a few drops of sanious serum, after which it becomes cold and insensible to the touch, and exfoliates off in pieces the size of a bean up to that of the palm of a man's hand; leaving a sore which may extend to the underlying muscles. Apart from this spontaneous, anæmic, cutaneous necrosis, the skin has a great tendency to form sores from continued lying down, and from other kinds of pressure, especially on the nose (where the nose-band comes), "girth place," edges of the lower jaw, bones, shoulders and orbital arches. The rapid disappearance of the cutaneous swellings is sometimes an unfavourable sign which may mean even death, when it is due to a sudden and abundant sero-hæmorrhagic extravasation in the intestinal mucosa with subsequent absorption of the subcutaneous infiltration, or when it is caused by a rapid rise of temperature, in which case the increased metabolism leads to the absorption of any nutrient material that may be present.

Swellings on the *limbs* render the gait awkward, prevent the animal from lying down and getting up, give rise to expressions of pain when the limbs are being bent, and often make it impossible for the horse to be moved from the spot he occupies. When the lower part of the head is considerably swollen, the opening of the nostrils becomes proportionately narrowed, and sometimes even completely closed, with the result of excessive dyspnœa, a wheezing form of breathing, and finally suffocation, unless the condition is relieved in time.

As regards the *digestive apparatus*, we have to point out that the mucous membrane of the oral cavity sometimes shows petechiæ, but rarely ulcers. At the commencement of the attack, food may be eaten fairly well for several days; although petechiæ may be present on the nasal mucous membrane, and swellings on the integumentum commune. Later on, mastication often becomes difficult and finally impossible by swelling of the lips and cheeks. With the appearance of fever, the appetite becomes disturbed and constipation sets in. Swallowing is frequently prevented by grave implication of the pharynx, on the mucous membrane of which, petechiæ and œdematous

an epidemic in Indian buffaloes where there were remarkable and extensive hæmorrhages of the skin and crater-shaped ulcers. Dettman observed a case of petechial fever in a goat.

The multiple hæmorrhages of the muscular system, which often occur in pigs, are not petechial fever; but are, according to Ostertag, lacerations of the fibres of the muscles caused during the transport of the animals. Ellinger assumes, in addition to this, an acquired hæmorrhagic diathesis as a predisposing cause. Bock reported a case of infection to a man from diseased horses.

Course.—The course of petechial fever is very atypical and irregular. In some mild cases, absorption of the hæmorrhagic and inflammatory foci takes place even within a week, without any grave complications; although in the majority of the mild attacks, the formation of hæmorrhagic infiltrations and swellings of the skin last from 8 to 14 days, after which, improvement gradually sets in. If, however, the hæmorrhagic infiltration of the skin and the mucous membranes is extensive, especially if the mucous membrane of the pharynx and intestines becomes involved, absorption of the inflammatory transudates will not usually take place earlier than in from 4 to 6 weeks, or even later. It leads sometimes to alternating metastasis in some of the internal organs (intestines, heart, lungs). A chronic course of petechial fever is also observed in isolated cases, when the disease continues with more or less virulence for some months. According to the observations of Kleinpaul the disease in a horse lasted even more than a year with prolonged intervals, during which it appeared healthy; in another case in the Bavarian cavalry it lasted five months before the horse died. Zschokke found, among 17 cases, that the disease lasted, on an average, 16 days. The shortest duration, which occurred in a fatal case, was 40 hours; the longest, 43 days. Cases have occurred in which the swellings of the skin and mucous membranes disappeared quite unexpectedly and without treatment. These cases of sudden improvement have been observed even in animals which have been given up by the veterinary surgeon who treated them. Death may ensue in a few days, in consequence of internal hæmorrhages, rapid sepsis, suffocation, paralysis of the small intestine, or when the attack has been the result of a grave primary disease. Gangrenous pneumonia (caused by foreign bodies) is particularly fatal. If death takes place after the petechial fever has lasted for a considerable time, its cause will generally be secondary septicæmia or pyæmia, the symptoms of which will be:

debility, high internal temperature, depression and diarrhœa. Ulcerating skin lesions require, as a rule, several months to heal. They may also cause cicatrical contractions, especially when they occur on the flexor surface of joints, producing, for instance, excessive uprightness of the pasterns. In such cases it is almost always better to kill, than to put the animal under a long course of treatment; for prognosis is very unfavourable.

Differential Diagnosis.—The respective course may run simultaneously with petechiæ on the nasal mucous membrane, and with circumscribed swellings of the skin (wheals). In horses, these diseases are chiefly strangles, anthrax, septicæmia, contagious pleuro-pneumonia, pyæmia, acute glanders, and erysipelas; and in cattle, quarter-ill, "deer and cattle disease," septicæmia puerperalis, other forms of septicæmia, malignant œdema, and poisoning by phosphorus and mercury. Consequently, it is not always easy to differentiate these diseases from petechial fever, especially at the commencement of an attack. The presence of diffuse swellings of the skin with extensive eruption of petechiæ, and the absence of the specific bacilli of anthrax, quarter-ill, malignant œdema, etc., will make the diagnosis positive in most cases. The same remark holds good in the differentiation of petechial fever from urticaria and simple cutaneous cellulitis.

Prognosis.—As the disease is liable to many complications and as its course is generally very tedious, our prognosis should be given with great caution. The mortality averages 50 per cent. In 17 cases observed by Zschokke 12 deaths occurred (70 per cent.). The following are unfavourable symptoms: extensive formation of petechiæ and inflammatory swellings; tendency to necrosis of the skin and mucous membranes; grave implications of the mucous membrane of the pharynx, larynx, and intestines; stenosis of the nasal cavities by swellings; high internal temperature; frequent pulse, exceeding 80 per minute; entire loss of appetite; rapid disappearance of the swellings of the skin; fœtid smell of the expired air; inflammation of the lungs; excessive weakness; inclination to decubitus; profuse diarrhœa; and great depression. We may take a favourable view of those cases in which the bodily condition is well sustained, the extent of the hæmorrhages and swellings slight, the appetite good, and

the internal temperature not much above normal. As we have already stated, severe symptoms are sometimes followed by unexpected amelioration and recovery. We should therefore no be too hasty in ordering the slaughter of horses which appear to be very gravely affected.

Therapeutics.—With regard to hygiene, our first care should be to provide a roomy loose-box in which the animal can freely move about; and we should remove the head-stall and surcingle. When difficulty in swallowing sets in, we should, if possible, give green food, or, failing that, mashes. Among the many medical agents which have been used to combat this disease, we may mention calomel, salicylic acid, sugar of lead, tannic acid, ergot of rye, extractum hydrastis, preparations of iron, camphor, quinine, arsenic, carbolic acid, iodide of potassium, creolin, lysol, ichthyol, oil of turpentine, sulphuric acid, and hyd ochloric acid. Some give boric acid (⅜ oz. in the drinking water) or cinchona bark (1¾ ozs. as an electuary). All these medicines produce at times a good effect; but in the large majority of cases they fail to favourably influence the disease. In France, petechial fever has recently been treated, with alleged success, with serum-therapy (anti-streptococcus serum according to Marmorek). Argentum colloidale is regarded by Dieckerhoff as a certain remedy for petechial fever, used as an intravenous injection (6 to 12 grains in a 1 per cent. aqueous solution). A number of observers have obtained very favourable results with this agent (Meissner, Röder, Richter, Kröning, etc.). Others consider it useless. Duschanek, for instance, had no favourable result from its use in five fatal cases, and cautions others against too sanguine hopes in this connection. Straube has ceased to employ argentum colloidale, as he has had to complain of five successive fatal results in spite of the employment of this remedy. The experiences at the Buda-Pesth School of Medicine have also shown that argentum colloidale is no specific against petechial fever. Huss is just as little convinced of its specific influence. In Dieckerhoff's Clinic, it appears that they have recently been convinced to the same degree of the inefficacy of this remedy, as instead of it the ichthargan recommended by Bass is now used for intravenous injections (Lange). Straube has successfully treated many horses with a 10 per cent. preparation of Jodvasogen (⅜ oz. per day, internally).

We may try to relieve the nasal dyspnœa in the first

place by passing a suture through the median alæ nasi, drawing it up and laying it over the middle line of the nose. Or we may follow Johne's advice and introduce metal tubes into the nostrils. In most cases, however, when this dyspnœa is complicated with laryngeal stenosis, tracheotomy will be our only available resource. We do not agree with the idea held by some that this operation should be performed earlier than necessity demands, so as to obviate the risk of the wound becoming gangrenous; because gangrene may supervene as a consequence of the accidental occurrence of septicæmia. Local treatment of the cutaneous swellings with astringents also appears to us to be usually superfluous. In particular, we have often applied Burow's solution (alum, 1 part; acetate of lead, 2 parts; water, 100 parts); but with varying results. We use aluminium acetate as an external antiseptic only when the swellings show breaches of continuity. In all other cases, we employ only inunctions of pure oil or paraffin ointment to lessen the tension. Inunctions that irritate or cause inflammation are decidedly objectionable. Like Röll, we have seldom found any advantage, but rather the contrary, from scarifications of the most swollen parts of the skin, with the object of obviating sloughing. On the other hand, Dieckerhoff reports favourably on the effect of deep incisions of the swellings. The treatment of the sloughing and ulcerating parts of the skin is purely surgical. Aruch recommends cold irrigations continued for several days as a good remedy for excessive swelling of the head.

The Treatment of Petechial Fever with Iodine.—Dieckerhoff has strongly recommended intratracheal injections once daily of ½ to 1 oz. of Lugol's solution (iodine, 1 part; potassium iodide, 5 parts; and distilled water 100 to 200 parts). The numerous reports in the literature of the last few years, on the effect of these injections of iodine, are very contradictory; in fact, while some praise them highly, others speak of them as absolutely inefficient, and even as injurious. Zschokke, who treated more than a dozen cases of petechial fever with injections of iodine, considers that their results were not so good as those which he had obtained with the remedial means he had formerly employed. Johne states that all the 3 horses which were treated with injections of iodine in Dresden died; out of 2 others which were treated with iodine and calomel at the same time, 1 died; and 1 horse which was treated exclusively with calomel recovered. In two cases, injections of iodine were followed by necrotic tracheitis and gangrene of the lungs. We ourselves have observed the occurrence of granular tracheitis and bronchitis as a result of these injections. Lemke and Buch report that horses died of 30 grammes (1 oz.) of the above-mentioned solution of

iodine. As about 50 per cent. of the cases of petechial fever recover without any medicinal treatment, and as apparently hopeless cases of this disease sometimes recover without treatment; we cannot view with favour the iodine method. Up to the present it would appear that the drawbacks in the employment of intratracheal injection of iodine in petechial fever are even greater than its advantages (as has also been demonstrated by Trinchera in the case of glanders). On these grounds we must recommend special caution.

DISTEMPER.

Distemper of dogs—Distemper of cats.

History.—Canine distemper has always been regarded as a very dangerous disease to dogs, and was described by various observers as distemper, canine plague, canine glanders, wheel-plague, catarrhal fever, etc. Laosson cites no less than 105 authors who have dealt with the disease, and states that it was known in the time of Aristotle. He considers that it was the canine epizoötic which raged in Bohemia during 1028. It is now the general opinion that distemper was imported to Europe for the first time about the middle of the 18th century from America (Peru, according to Heusinger). It was first brought to Spain, from which it was taken to France, Germany, and other countries. It is said to have reached France about the year 1740; Germany about 1748; Italy about 1764; England about 1760; and Russia about 1770. At the present time it is spread all over Europe.

Formerly distemper was regarded as a disease resembling plague and strangles; also, as a nervous disease, or a nervous febris mucosa; others regarded it as typhoid, and even as typhus identical with that of man. Many old authors considered it to be true small-pox, with which opinion Trasbot agrees; and numerous unsuccessful experiments were made with vaccine as a preventive against it. This hypothesis has been refuted by the recent experiments of Dupuis, who in no instance was able to produce immunity against distemper by vaccinating young dogs. Many supposed it to be a disease of development or a constitutional affection, even the sodium-inanition of the body has been looked on as a cause.

It was recognised by Waldinger, von Gemmeren, Delabère-Blain and others at an early period, that distemper was spread by an infective agent; and consequently numerous artificial

transmission experiments were made with its virus; the first successful experiments being those by Renner and Karle. Trastowo proved by his experiments, that young dogs which had not suffered from the disease were susceptible to it; that it could be transmitted directly and indirectly; and that old dogs could be infected by it. Trasbot was at first unsuccessful with his inoculation experiments; but later on he was able to transmit it by inoculation to dogs, from 13 days to 3½ months old, by introducing the nasal discharge mixed with the contents of the vesicle, into small incisions on the abdominal walls, with the result that the first symptoms of the disease appeared in 8 days. He also produced infection through cohabitation, which experiment was confirmed by Venuta, who further proved that the contagium was fixed as well as volatile, and that it had sufficient vitality to bear drying in the air up to a certain degree. Venuta found that the period of incubation varied from 4 to 6 days. Krajewski inoculated 36 dogs, of which the greater number remained unaffected; because, as he surmises, they had passed through a previous attack of distemper. He found that the period of incubation varied from 4 to 7 days, and that an increase of internal temperature was the first symptom. An exanthema seldom appears, and only in severe cases. In very mild instances, recovery takes place as early as 6 or 8 days. One attack generally confers immunity. The contagium is contained in the respective discharges from the nose and eyes, and in the blood. Although it is not destroyed by drying, or by freezing down to -4° F.; its action is weakened by being kept for months in a dry condition. As distemper by inoculation has a mortality of only 10 to 15 per cent., Krajewski recommends inoculation as a prophylactic measure. Laosson made, on dogs and cats, 98 inoculation experiments, which positively demonstrated the contagious nature of distemper. He proved that the disease was identical in both of these animals, and that it can be reciprocally transmitted. He further showed that young cats and dogs became infected almost without exception; that mature and old cats and dogs are less susceptible; that the nasal discharge loses its virulence in 14 days; that the contents of the pustules are ineffective; and that the period of incubation varies from 4 to 7 days. Konhäuser, whose inoculation experiments had a negative result, is of opinion that the contagium is also present in the milk of bitches suffering from distemper.

Etiology.—Distemper is a contagious, infective disease, the virus of which has not yet been positively demonstrated. We have not yet been able to obtain pure cultures and their successful transmission. We only know that the contagium is fixed as well as volatile. It is evidently much easier transmitted by cohabitation than by inoculation, and is probably, as a rule, taken from the air during inspiration. Dogs become most frequently affected during the first year of life. Among the 1,378 canine patients which were treated for distemper during one year at the Berlin Veterinary College, 927 (two-thirds) were under 12 months; 269 (one-fifth) were under 2 years; and 182 (one-seventh) over 3 years old. In rare cases, very old dogs may become infected. Puppies only a few weeks old may also suffer from this disease. Contrary to the statements of Krajewski, we have seen an outbreak of distemper in a whole litter of puppies which were between 2 and 3 weeks old. Although one attack generally confers immunity for a considerable time, some dogs become infected several times. We have, for instance, treated the same dog for distemper 4 times in a year.

The chief *predisposing cause* of distemper is chill, brought on, for instance, by exposure in the open air, particularly at night and during cold and wet weather; injudicious washing and bathing; etc. Chill facilitates the penetration of the virus by weakening the vital forces, or by producing a simple catarrh of the respiratory mucous membranes. A cold of itself can never produce distemper. High-bred, pampered, weak, recently imported, and unacclimatised dogs are specially predisposed to this disease, the occurrence of which is favoured by an unnatural system of feeding and rearing. The old prejudice against meat for the food of young dogs is often the indirect cause of distemper. We may point out that bread weakens the health of dogs, and that meat is the only appropriate food for carnivora. According to the experiments of Bischoff and Voit, dogs which are exclusively fed on bread get into a wretched plight; and cats even die. Progeny predisposed to distemper is likely to be obtained from weakly bitches, and from those which have to bring up an excessively large number of puppies. Considerable hæmorrhage, for instance, by cropping the ears and tail, and rickets, also have a predisposing influence. The supposition that certain breeds become more frequently affected with distemper than others, is probably erroneous; the more likely cause of this apparent

susceptibility being the fact that such breeds are more numerous than others. Thus we find in Berlin that more than one-third of all dogs suffering from distemper are pugs; apparently because this breed is at present fashionable, and not because it possesses any special predisposition to distemper.

Bacteriology.—The exact nature of the agent of the infection of distemper has not, up to the present, been fully determined. The views of authors in this respect widely differ. Semmer and Laosson found in the blood of affected dogs, a few hours after death, exceedingly slender, small bacilli, which they consider to be the true microbes of distemper. Rabe found in the purulent contents of the pustules, in the nasal discharge, and in the conjunctival secretions of dogs suffering from distemper, bacteria which he regards as the infective agent of distemper. These micro-organisms consisted of globules, the size of which was uniform; but they were so minute that they could hardly be measured. They sometimes lie together in small heaps, or are connected with one another in twos and fours (like sarcinæ), or lie in rows of four or five like a string of pearls. They can be stained a dark-blue with methylene-violet. During convalescence, they disappear completely from the nasal discharge, and vary in number proportionately to the severity of the attack. Friedberger confirmed Rabe's statement, but leaves the question of the specific nature of these micro-organisms unanswered for the present. Also, Krajewski noticed the presence of micrococci. Mathis found in the fluids of the body, tissues, sputum, and pustules of dogs suffering from distemper, a specific diplococcus, which he cultivated in neutral or slightly alkaline broth, obtained pure cultures up to the seventh generation, and made successful inoculations with them. The symptoms which appeared after the inoculations, agreed in many respects with those of distemper. For instance, there was generally a rapid rise of temperature, and pustules appeared on the site of the inoculation or over the whole body. Very young animals frequently died in consequence of this inoculation, which conferred immunity on the survivors. Jacquot and Legrain found in the pus of the pustules numerous motile micrococci, which were from 0·6 to 0·8 μ in diameter, and which, by uniting, formed diplococci. Inoculations with their cultivations produced local pustules, but no distemper. Marcone and Meloni obtained the same result with the micrococci which they found, and which possessed great similarity to staphylococcus pyogenes aureus. Galli-Valerio found in the lungs, brain, spinal medulla and pustules of dogs suffering from distemper, ovoid bacteria, which were 1·25 to 2·5 μ long, and 0·3 μ broad, and showed characteristic cultivations. Their inoculations produced in dogs symptoms of distemper, especially those of a nervous type. Babes and Barzanesco (1896) have isolated a bacillus in two cases; of 9 dogs inoculated therewith, 7 succumbed to distemper in 10 to 18 days. Millais (1896) isolated a micro-organism similar to the bacillus of pneumonia; also micrococci. Rubbing of a mixed culture into the nose of a young dog produced a mild form of distemper. Jensen (1896) found that the pneumonia of distemper was produced by streptococci. Taty and Jacquin (1898) found, in the nerve-distemper, diplococci were present in the central nervous system, which

they regarded as the causative agent of the disease. Jess (1899) has cultivated a bacillus of 1.8–2.3 μ in length, and 0.6–0.9 μ in breadth, which stained by the Gram method, formed a dull-grey film on agar, and a white one on potato; and was found to be pathogenic to guinea-pigs, cats and dogs. He considers that this bacillus is not identical with any of the micro-organisms which have hitherto been observed associated with distemper, and maintains that he has produced distemper in dogs by injection of pure cultures of it. Petropawlowsky (1899) claims the discovery of a specific bacillus; which, nevertheless, according to Mari, is but a bacillus coli. Lignières (1900) reckons distemper among the group of Pasteurellar affections, and claims to have isolated a Pasteurella canina from the cultures of which Phisalix has prepared an immunising serum; which was, nevertheless, found by Copemann (1900) in many cases ineffective against inoculation-matter prepared from the parasites alleged to be productive of distemper (Hobday, Sewell, etc.).

Schautyr tried to prove, in a bacteriological work published in 1892, that canine distemper should be divided into three different diseases, which are respectively produced by three morphologically, biologically and pathogenically different micro-organisms. He, however, states that these three forms of distemper are clinically and anatomically so much alike, that they can be distinguished from each other only by an exact bacteriological examination. He named these three forms: "abdominal typhus," "true canine distemper," and "typhoid." We do not feel inclined to give up the clinical unity of canine distemper for such bacteriological problems. The same view holds with regard to the separation, advocated by Mégnin, of distemper into two groups (strangles and distemper). Finally, Cadiot and Breton (1901) have described, as a special discovery, in addition to the distemper characterised by the presence of the exanthema, the existence of an infective broncho-pneumonia as an independent disease.

Occurrence.—Cats, wolves, foxes, jackals, hyænas, and monkeys become affected with distemper as well as dogs, which suffer from it sporadically, enzoötically, and even epizoötically. It is almost always present in large towns, in which it is the most common canine disease, and is particularly frequent during certain years. Among the 70,000 dogs which were suffering from internal and external diseases, and which were taken to the Berlin clinic during the years 1886 to 1894, the numbe of cases of distemper was over 11,000 (one-sixth of all cases). The proportion was about the same in Munich; for among the 2,300 dogs which were received in the Munich clinic during 14 years, and which were suffering from internal diseases, 650 were suffering from distemper. This disease can also spread with great rapidity in the country. Jonsson states that distemper raged so violently in Iceland, that there were no dogs seen in large districts of that country. According to our experience, the majority of outbreaks of dis-

temper occur during the summer months. Warmth seems to greatly favour the development and transmission of the contagium.

Symptoms.—The symptoms of distemper vary greatly, and are chiefly those of an infectious catarrh of the mucous membranes of the eyes, and respiratory and digestive organs. These catarrhal symptoms may become complicated with symptoms of severe disturbance of the brain and spinal cord, characteristic exanthema of the skin, and frequently with catarrhal pneumonia. The diversity in the course of the disease has led to the division of distemper into various forms, according to the localisation of the disease, such as catarrhal, nervous, exanthematic distemper; or distemper of the eyes, gastric distemper, pulmonary distemper, etc. These varieties often occur in a pure form, that is to say, without any other complication. Thus, the symptoms of an attack of distemper may be essentially those of an infection of the brain, intestines, or eyes, or of an eruption of the skin. As a rule, however, several organs are simultaneously affected. Owing to the want of uniformity in the course of the disease and number of its complications, we can describe only the most important affections of the individual organs.

1. *Initial symptoms.* Distemper is usually first manifested by constitutional disturbance, such as: decreased vivacity, dainty and small appetite, bad temper, trembling and shivering, bristly condition of the coat, warm and dry state of the nose, and tendency to become easily fatigued. In inoculation experiments, there is a considerable increase in the internal temperature, which, even during the period of incubation, rises to 104° F.; and, at the commencement of the attack, up to 106° F. in slight cases, and to 108° F. in severe ones. Owing to the fact that dogs are put under treatment, as a rule, only when the disease has become well-developed, few exact observations have been made on the state of the temperature during the initial stage. It is, however, not improbable that distemper, like other acute infective diseases, commences with high temperature, which lasts only for a short time.

2. *Eye symptoms.* In the large majority of cases, conjunctivitis is the primary local symptom observed in the eyes, in the form of a serous conjunctivitis, which afterwards becomes purulent. Tears flow from the eyes, and photophobia is present. The mucous membrane of the eyelids becomes highly

congested and swollen, and the eyelids turgid. The discharge from the eyes, which is at first serous, soon becomes mucous and purulent. The exuded matter consists of pasty mucus, or dirty yellow pus, which looks like cream or butter. This exudate collects under the lower eyelids, chiefly at the inner canthus of the eye, and soils the edges of the eyelids, upon which it frequently dries into crusts and causes the eyelids to adhere together, particularly during night. Lesions and ulcers form on the cornea as consequences of the action of the accumulated and decomposing pus; by the patient wiping and rubbing its eyes with its paws; and by defective nutrition. The epithelium of the cornea sometimes suffers, more or less, from shallow, flat lesions, which give the surface of the cornea a rough and uneven appearance; or from smaller but deeper ulcers, especially towards the centre of the cornea. These ulcers, which are often not larger than the head of a pin, are funnel-shaped and penetrate downwards in a straight direction; their base frequently becomes coated with a pus-like material; and they heal by proliferation of vessels from the edge of the cornea. In many cases, during their further course, Descemet's membrane protrudes, the cornea becomes perforated, prolapse of the iris ensues, and staphyloma is set up; the usual result being that cicatrisation takes place with the formation of permanent white spots (leucoma) and black pigmentation of the cornea, but rarely with suppurating panophthalmia. Vesicles, in very rare instances, precede the ulcers on the cornea.

In other cases the cornea, generally of both eyes, becomes affected with a diffuse, parenchymatous keratitis, which renders it, to a considerable extent, opaque, and gives it the appearance of ground glass. These extensive opacities sometimes become developed in a comparatively short time. This affection of the cornea (so-called "distemper of the eyes") is frequently the only symptom of distemper which is present, with the exception of the high temperature. The surface of the implicated cornea is smooth and shining, and the conjunctiva is quite intact. Internal inflammation of the eye, as for instance, an exudative iritis with a fibrinous or purulent exudation into the anterior chamber of the eye, is rarely seen.

3. *Digestive symptoms*, the chief of which are loss of appetite, vomiting, well-marked congestion and dryness of the oral mucous membrane, abnormally great thirst, constipation at first, and diarrhœa later on, in which case the fæces, as a rule, are very

fœtid, often slimy and frothy, and even bloody (hæmorrhagic intestinal catarrh). Jaundice rarely occurs. The urine frequently contains albumen—especially when the patient is weak, or is in an advanced stage of the disease—and biliary pigments; but rarely hæmoglobin. In many instances, these are the only symptoms present.

4. *Respiratory symptoms.* The first symptoms are those of nasal catarrh, such as a nasal discharge which is serous at first, and mucous or purulent later on; followed by sneezing, panting and nasal pruritus, which causes the animal to wipe its nose with its paw and to rub it on its fore-legs. The pus discharged from both nostrils is often very copious, is sometimes mixed with streaks of blood, and varies in colour from dirty yellow to dirty green. Later on, it may become fœtid and even watery. Ulcers are often seen on the nasal mucous membrane. The copiousness of the secretion points to participation of the accessory nasal cavities. The nose often becomes very dry and chapped.

Laryngeal catarrh generally accompanies nasal catarrh, and manifests itself by a cough, which comes on in paroxysms, is at first hoarse and dry, and later on moist and accompanied by a discharge of phlegm. This cough excites the animal to vomit. The catarrh spreads from the larynx to the trachea and bronchi. The resulting bronchitis is followed by increased rate of breathing and manifests itself by cough; hoarse, sharp, vesicular respiratory murmurs; and rhonchi. The mucous membrane of the smaller bronchi is frequently catarrhally affected (bronchiolitis or capillary bronchitis), under symptoms of increased respiratory frequency; difficulty in breathing; feeble, harassing cough, which may also be set up by percussion of the thoracic walls, by the animals getting up, and by their being taken out of their kennels, and which the patients try to suppress; humming and whistling sounds; and dry or moist, crackling or fine râles. Young and weak animals which cannot remove the accumulated phlegm from the bronchi by coughing, become affected with catarrhal pneumonia on account of this excretion becoming drawn into the alveoli. This pneumonia can be recognised by the great increase in the internal temperature; excessive dyspnœa (pumping up the air and puffing out the cheeks); greater or less disappearance of the vesicular respiratory murmur on circumscribed parts of the thorax; irregular and weak dulness of the percussion-sounds, which may, however, be absent in the case

of the inflammatory focus occupying a median position; occasional tympanitic percussion-sound; and by the bronchial breathing, which is sometimes audible. The cough is in this case very dull and weak, and the nasal discharge is often fœtid. With approaching paralysis of the heart, we may have symptoms of œdema of the lungs, namely: excessive dyspnœa, crackling râles, bubbling rhonchi, tympanitic percussion-sound, and gradual decrease in the frequency of respiration.

5. *Nervous symptoms.* Distemper often begins, especially in anæmic animals, with great depression and dulness. Strong animals, however, more usually exhibit symptoms of acute hyperæmia of the brain, such as excitement, high temperature of the head, restlessness, yelping, and even attacks of fury, which give way, later on, to manifestations of cerebral pressure (stupor and a stretched-out condition of the head and neck). These cerebral disturbances may be the only symptoms present in the nervous form of distemper, which at certain times is pretty widely spread. Also, tonic and clonic spasms frequently appear, either generally or locally, and are often confined to particular limbs, which swing backwards and forwards as if affected by chorea. These movements may last even for days, or the patient may have spasms of the muscles of the head, particularly of the facial muscles which receive their nervous supply from the facialis, and muscles of mastication which are supplied by the third motor branch of the trigeminus, in which case, spasms also appear on the lips, cheeks, eyelids, temporal muscles and masseters. Spasm of the masseters often produces a persistent and apparently automatic opening and closing of the mouth. Convulsive contractions of the panniculus, especially in the region of the back, may be seen; and also extremely severe epileptiform convulsions, which are sometimes restricted to the head and neck, but at other times are spread over the whole body. The sick dog, first of all, becomes restless and excited, stares stolidly at objects around him, shakes his head, and runs aimlessly about. Subsequently he is attacked either by spasms of the muscles of mastication, in which case he froths at the mouth, the head and neck are drawn backwards and sideways, and the muscles of the face are convulsed; or he falls down as if from epilepsy, barks, yelps, becomes unconscious, and exhibits tonic and clonic spasms in almost all the muscles of the body, in which case, the sphincters of the anus and bladder become relaxed, and consequently fæces and urine are involuntarily passed. There is a gradual return

of consciousness in a short time, usually as soon as 30 to 60 seconds, and the dog manages to get up, although he is very weak. Such an epileptiform attack may pass directly into long-continued coma. Circular and rotatory movements are somewhat rare.

Paralysis also appears, usually as a sequel to the convulsions; although it may come on simultaneously with them. It seldom occurs at the beginning of the disease. It may be confined to certain groups of muscles, as for instance those of particular limbs; although it very often affects the whole of the hind-quarters, or even the entire body in the form of paresis, especially of the motor nerves, combined with excessive muscular weakness. The sick dog staggers and his hind-quarters sway from side to side, or he becomes incapable of supporting himself on his hind-legs. Frequently, he knuckles over on all four legs, and in severe cases is unable to stand. Permanent paresis of the hind-quarters with paralysis of the bladder and rectum is a frequent sequela of distemper. In some cases, we have noticed paralysis of the muscles of the tongue, which consequently hung loosely out of the mouth, and prehension of food was greatly hindered. During the course of distemper, the following complications have been observed: deafness; amaurosis; cataract in very rare cases; hemiplegia laryngis (roaring); hydrorachis; loss of voice (aphasia), smell and memory; and complete idiocy (hydrocephalus internus). According to our experience, the most common complications are: amaurosis with optic atrophy and chronic hydrocephalus. In a few cases, we have seen unexpected recovery from such amaurosis.

6. *Cutaneous symptoms.* A characteristic pustular exanthema is frequently observed on the inner surface of the thighs and abdomen, and may be the only symptom of distemper in abortive cases, which are the mildest kind of this disease. It first appears in the form of minute red spots, which after twenty-four hours develop into miliary nodules that are surrounded by a red ring. These nodules change into vesicles and pustules, which vary in size from a lentil up to a pea or even a bean, and dry up either into a yellow-brown crust, or burst, and leave weeping, sore spots. Healing takes place with desquamation of the epidermis after about 8 days; leaving bright pigmented, pale-reddish spots, which persist for some time. Generally, there are only a few pustules present. The exanthema may also spread, in the form of a scabby eczema,

over the whole body, to the membrane of the external auditory meatus, and less frequently to the mucous membranes of the mouth and eyes. In such cases, the exhalations from the body have a bad smell, and some of the hair falls out. This eruption, contrary to that of sarcoptic mange, is accompanied by only slight pruritus. Intense catarrh of the prepuce may appear simultaneously with the exanthema. An eruption of wheals (urticaria), spreading over the whole body, has been observed in a few cases.

7. *General symptoms.* The internal temperature, which is usually very atypic, is high during the initial stage, and when local manifestations appear in the lungs, intestines, etc. It often falls below normal with remarkable rapidity towards the approach of death and may be as low as 99° F., 94° F., or even 90° F. If the disease runs a protracted course, the patient becomes emaciated; the hind-quarters waste away; the ribs show through the skin; the hair stands on end, and loses its lustre; the bodily exhalations have a very fœtid smell; the eyes are deeply sunken; the mucous membranes are pale; and the patient is weak, staggers when walking, or maintains a recumbent position while in a state of coma.

Distemper of Cats.—Krajewski proved, by the experimental transmission of the distemper of cats to dogs, and *vice versâ*, that the two diseases are in every respect identical. Feline distemper may occur enzoötically. Its symptoms are precisely the same as those of canine distemper. Nervous attacks, however, appear to be rarer in cats than in dogs, and may be entirely absent. The initial symptoms consist of loss of appetite, retching, vomiting, depression, etc., followed by suppurating conjunctivitis, purulent nasal discharge, sneezing, coughing, snorting, difficult breathing, diarrhœa, screaming, weakness, anæmia, and, towards the end, symptoms of coma with a fall of internal temperature. There seems to be no exanthema. On *post-mortem* examination, we find changes similar to those in dogs. Contrary to the statements of other observers, we have found on several occasions, especially in cats, extensive croupous pneumonia. With regard to therapeutics, we would advise exactly the same treatment for cats as for dogs, with the exception of the omission of carbolic acid, which has a very poisonous effect on cats. The doses appropriate to these animals are about the fifth of those usually given to dogs.

Course and Prognosis.—Distemper has more varieties than almost any other disease. Sometimes it is limited to a pustular exanthema, a parenchymatous keratitis, a mild nervous affection, or a slight catarrh of the mucous membranes of the head or digestive organs. In such, so to speak, abortive

cases, a recovery may take place as early as 8 or 10 days; although the disease usually lasts 3 or 4 weeks. With severe complications, especially with those of the nervous system, distemper assumes a protracted course, and is frequently followed by sequelæ, such as paralysis and convulsions, which may persist for months, and even during the whole life. In the lungs, chronic, destructive processes which resemble phthisis and which predispose to tuberculosis, are associated with catarrhal pneumonia. The average mortality is from 50 to 60 per cent. Among unfavourable factors we may mention extreme youth, high-breeding, weakness, anæmia, extension of the process to several organs, convulsions, paralysis, pneumonic attacks, profuse diarrhœa, continued high temperature, emaciation, exhaustion, a depraved condition of the excretory organs, perspiration, and abnormally low temperature of the body. Death takes place from paralysis of the brain (which may have a fatal termination even in a few days), œdema of the lungs, septicæmia, pyæmia, or general exhaustion. Mature age, common breed, and restriction in the spread of the morbid process are favourable conditions. Our experience does not agree with the opinion expressed by some, that the fact of certain forms of distemper being complicated with the above-described exanthema of the skin is prognostically unfavourable.

Differential Diagnosis.—In making our diagnosis, we have frequently to decide the question whether we have to deal with distemper, or with a simple primary catarrh, such as that of the eyes, nose, lungs, stomach, or intestines. To this, a precise answer cannot always be given; because distemper frequently appears as an affection of merely one organ. In general, however, we obtain data sufficient for our purpose from the epizoötic character of the disease, youth of the patient, high fever, simultaneous implication of several organs, and unfavourable course of the malady. The demonstration of the pustules of distemper is of great diagnostic importance. The symptoms of cerebral irritation which occur at the beginning of the disease may give rise, especially in the minds of laymen, to the suspicion of rabies. The characteristic aggressive behaviour of animals suffering from rabies is, however, absent from distemper, the further course of which soon settles the matter. The eruption of distemper may resemble that of mange by its becoming greatly extended. The mild

character of the pruritus, the presence of pustules on the hypogastrium and inner surface of the thighs, the rapid spreading of the exanthema over the whole body, and the development of other symptoms of distemper, render the differentiation easy. The exanthema of distemper and that of mange, however, often occur simultaneously in the same animal, in which case certain precautions are necessary for making a correct diagnosis. Epileptiform attacks of distemper are essentially distinguished from true epilepsy by their acute character.

Anatomical Conditions.—The anatomical changes in the *respiratory system* are those of rhinitis, laryngitis, bronchitis, and catarrhal pneumonia. The nasal mucous membrane is either very pale, or greatly congested, swollen, and covered with a thick, purulent greyish-green or dull-red inflammatory exudate which is mixed with coagulated blood, and which collects chiefly between the lamellæ of the turbinate bones and in the frontal sinus. Hæmorrhagic ulcers are sometimes present. The mucous membrane of the larynx and bronchi is also highly congested, swollen, infiltrated with hæmorrhages, covered with pus, and sometimes shows catarrhal ulcers. The large bronchial tubes often fail to exhibit changes which might have been expected during life; for the hyperæmia and the swelling of the mucous membrane subside after death. The smaller bronchi are, on the other hand, frequently filled up with dirty-grey and even bloody viscid pus. There are spots of congestion on the surface of the lungs, some parts of which may contain but little air, may be entirely free from air, or may contain more air than usual. The catarrhal inflammatory foci of the lungs are characterised by their hardness, absence of air, prominence, and dull-red or copper colour. Their section has a smooth or, in rare cases, a slightly granulated surface, which, when pressed, gives off an opaque, chocolate-coloured fluid that later on becomes dull-red or pale green. In very young animals we may observe the infiltration, into the parenchyma of the lungs, of a fibrinous exudate, which is very soft, and which readily liquefies. This croupy hepatisation frequently has a lobar extension. The inflamed parts of the lungs are sometimes densely studded with numerous suppurating foci, which vary in size from a poppy-seed to more than that of a hemp-seed, or are diffusely infiltrated with pus. Those portions of the pleuræ which cover the affected parts often undergo inflam-

matory changes. The bronchial glands are swollen and infiltrated with serous fluid, or with pus.

In the *digestive system*, we find the mucous membrane of the stomach and intestines, especially that of the small intestine, congested and swollen, covered with tough, glassy mucus and often infiltrated with hæmorrhages. In other cases, it is very pale, swollen, easily torn, and has a creamy covering. Frequently, the contents of the intestine are sanious; and the mesenteric glands œdematously swollen. In very rare instances abscesses of the lymphatic glands are found in the abdominal cavity.

In the *brain*, we often find signs of œdema, such as: anæmia; a relaxed and soft condition of the brain substance; a watery lustre on the surface of sections; flattening down of the convolutions of the brain; and serous effusions into the lateral ventricles and subarachnoideal spaces. In a few cases, we meet with signs of purely venous cerebral hyperæmia, as for instance, great congestion of all the sinuses, venous plexuses and vessels of the pia, and the appearance, on the section surfaces of the brain, of numerous red blood points, which can easily be wiped off. Kolesnikoff found microscopically the brain substance, especially the walls of the vessels, infiltrated with leucocytes; and Krajewski noticed dilatation of the vessels, cellular infiltration of their walls, filling of the perivascular spaces with lymphoid cells, and immigration of lymphoid cells into the stroma of the brain and into the protoplasm of the ganglionic cells. The changes in the spinal cord, which are not well-marked, consist chiefly in anæmia and slight œdema, especially in the lumbar region. Mazulewitsch, whose observations have been confirmed by Dexler, states that, in acute paralysis, we can find changes of the walls of vessels with an albuminous exudate along the vessels and in the interstitial tissue of the grey matter of the spinal cord; and in chronic distemper, a chronic, localised interstitial myelitis with partial atrophy of the cord. Hadden found groups of emigrated blood-corpuscles in it. According to Trasbôt, the spinal cord and its membranes in severe cases are often considerably injected with a sero-fibrinous exudate in and under the arachnoid, and even into the substance of the spinal cord. Hydrorachis may become developed in chronic cases.

Among other changes, we may mention: decrease in the total quantity of the blood of the body, combined with a certain degree of hydræmia: opacity and fatty degenerations of the

epithelium of the liver and kidneys; dull yellow discoloration of the muscular tissue of the heart in consequence of dust-coloured cloudy swelling and fatty degeneration of its fibres; œdematous swelling of the lymph glands; tarry and greasy condition of the blood (in complications with septicæmia); and capped elbow and capped hock from decubitus.

Therapeutics.—The treatment of distemper is essentially symptomatic. There are, however, medicines which are able to subdue, *in loco affectionis*, the contagium of distemper, after it has entered the body. Of such medicines, calomel is a good instance. According to our experiments, this drug can be used with advantage in every case in which the digestive organs have been the starting point of the infection. We obtained, especially in the initial stage of the so-called gastric form of distemper, good results from its administration two or three times a day, in doses of ¾ grain. Calomel acts as a disinfectant of the mucous membrane of the stomach and intestines. The same may be said of the disinfection of the respiratory mucous membrane by means of inhalations of creolin (½ to 1 per cent.), which have an equally good effect in the bronchial form of distemper. Our success in the treatment of the bronchial and pulmonary forms of distemper has been greatly enhanced by the adoption of creolin as a local disinfectant of the respiratory mucous membrane; although in this disease we are often compelled to use a purely symptomatic treatment, which varies according to the localisation of the malady.

1. When the *eyes* are affected, their treatment has to be carried out according to the rules of ophthalmology. A solution of ½ to 1 per cent. of creolin is an admirable application for suppurating conjunctivitis and ulcers of the cornea. In the former affection, good results may be obtained with a ½ to 1 per cent. solution of sulphate of zinc, or in obstinate cases, with a ½ to 2 per cent. solution of nitrate of silver; and in the latter, with a 2 per cent. solution of boric acid or quinine. We may treat parenchymatous keratitis with ½ per cent. solution of sulphate of atropine. A 1 to 2 per cent. solution of hydrochlorate of cocaine (5 minims to be dropped into the eyes every 3 hours) may be employed to combat the abnormal sensitiveness of the mucous membrane of the eyelids, which causes the patient to wipe and rub his eyes, and which, thus, often induces the formation of purely traumatic ulcers. In cases of blenorrhœa, we may cauterise the inflamed mucous membrane with a point

of pure or diluted nitrate of silver, with one of bluestone, or in very obstinate cases, with the thermocautery after a previous application of cocaine. Opacities of the cornea of some standing may be more or less removed by rubbing them with red precipitate ointment (1 to 20). Staphyloma and entropium require surgical treatment.

2. The affections of the *digestive apparatus* may be treated in different ways. An emetic (·15 to ·075 grain = $\frac{1}{6}$ to $\frac{1}{12}$ grain of apomorphine hydrochlorate in from 15 to 75 min. of water, subcutaneously) sometimes renders good service at the commencement of the disease. As a stomachic, we can recommend hydrochloric acid in doses of a drop by itself in water, or combined as follows: hydrochloric acid 1 part, tincture of gentian 4 parts, water 60 parts; half or a whole teaspoonful to be given 3 times a day. We may also administer tinctura rhei aquosa and vinosa (in doses of a teaspoonful); decoctions of cinchona bark (1 to 15, a tablespoonful at a time, by itself or combined with hydrochloric acid); bismuthi subnitras (1½ to 3 grains); extract of meat (½ to 1¼ dr.); wine, quinine wine, condurango wine (from a teaspoonful to a tablespoonful), etc. Opium with small bits of ice is indicated in persistent vomiting. Diarrhœa may be treated with tinct. opii, 7½ to 30 mins.; powdered opium, 1½ to 4½ grains; tannin, 3 to 7½ grains; and in very obstinate cases with nitrate of silver, ⅓ to ¾ grain in pills. In dysenteric diarrhœa we give opium with mucous remedies, as for instance, tinct. opii 14 parts, acaciæ gummi 15 parts, aqu. fœniculi 300 parts, 3 times daily a teaspoonful or a tablespoonful.

3. In affections of the *respiratory organs*, we should first of all employ the remedies which we have mentioned for the relief of laryngitis, bronchitis, and catarrhal pneumonia. For cough, we get good results with morphia and codeine, which we may prescribe as follows: R. morphinæ hydrochloras 1½ grains, aqu. amygdal. amar. 3 drs., aqu. destill. 5 ozs. 2 drs., M. a teaspoonful or a tablespoonful 3 times daily. Or: codeinæ phosphor. 4½ grains, syrupi althææ 3 drs., aqu. fœniculi 5 ozs. 2 drs., M. a teaspoonful or a tablespoonful every 3 hours. Besides this we recommend moist, warm fomentations over the larynx and thorax. The affection of the bronchi requires the employment of expectorants, such as apomorphinæ hydrochloras ($\frac{1}{6}$ grain *pro die per os*); tartar emetic (only for strong animals) and sul-

phurated *antimony* ($\frac{1}{3}$ *to* $\frac{3}{4}$ *grain*); chloride of ammonium ($1\frac{1}{2}$ to $7\frac{1}{2}$ grains); vinum antimoniale (in doses of a drop); infusum senegæ (a teaspoonful up to a tablespoonful); and ipecacuanha ($\frac{1}{3}$ to $\frac{3}{4}$ grain), etc.

4. For spasms caused by disturbance of the *central nervous system*, we prescribe potassium bromide and sodium bromide (1 to 25 of water, a teaspoonful or tablespoonful, 3 times daily); sulphonal (15 grains to 1 dr.); and chloral hydrate ($\frac{3}{4}$ to 75 grains) in mucilage; or subcutaneous injection of morphia ($\frac{1}{3}$ to $1\frac{1}{2}$ grains). We treat paralysis and weakness by stimulants, such as coffee (5 to 10 per cent. infusion), coffee with brandy, beef tea, extract of meat, wine, camphor, (spirit. camphor. or linimentum camphoræ $1\frac{1}{4}$ to $2\frac{1}{2}$ drs. rubbed into the skin); ether (15 minims every hour, subcutaneously); caffeine ($7\frac{1}{2}$ to 30 grains subcutaneously); hyoscine ($\frac{1}{13}$ to $\frac{1}{3}$ grains subcutaneously); atropine $\frac{1}{6}$ to $\frac{3}{4}$ grain); veratrine ($\frac{1}{65}$ to $\frac{1}{13}$ grain); strychnine ($\frac{1}{65}$ to $\frac{1}{22}$ grain subcutaneously); and electricity (Spamer's machine).

5. The *fever* (rise in the internal temperature) should be specially treated only when it attains a great height, or when by its persistent presence it threatens to become dangerous to the more important organs of the body, especially the heart. The initial fever, and any slight rise of temperature, say up to 104° F., require no antipyresis. As febrifuges we prefer antipyrine ($7\frac{1}{2}$ to 30 grains) and antifebrine ($3\frac{3}{4}$ to 15 grains); because they reduce the temperature with great certainty and in a short time ($3\frac{3}{4}$ to 15 grains every hour until the temperature becomes normal). The effect of quinine is not so certain as that of antipyrine or antifebrine.

6. The *skin* affection requires little or no treatment. Moist sore places may be dried with powders, such as 1 part of oxide of zinc to 10 of talc. The application of glycerine to the crusts of the dried-up pustules will hasten their removal.

Finally, for the treatment of distemper in the initial stage, subcutaneous injections of iodine tri-chloride ($1\frac{1}{2}$ drm. of a solution of 1 : 2000) have been recommended (Buda-Pesth School of Medicine).

As regards diet, we should give easily digested nutritious food (the best kind of which is raw meat minced or scraped), milk, and beef tea. Prophylaxis demands that the affected animals should be segregated from the healthy. The results of the value of protective inoculation (anti-distemper serum, immunising serum of Phisalix) are very contradictory.

The Canine Plague of Stuttgart.—Under this name Klett has described an infectious disease which first appeared in Stuttgart in the summer of 1898. It was regarded as essentially consisting of a hæmorrhagic gastro-enteritis, complicated with an ulcerative stomatitis and entirely distinct from distemper. This disorder had, by the end of 1890, become widely distributed: in Frankfort-on-the-Main (Scheibel), Munich (Albrecht), Dessau (Richter), Magdeburg (Grundelach), Zürich (Zschokke), Vienna (Tremmel), and also in other localities (Hamburg, Wiesbaden, Lössnitz, Edinburgh, Brighton). Its symptoms are a sudden attack of persistent vomiting, total loss of appetite, with extreme lassitude and languor (similarity to poisoning). From 3 to 5 days these symptoms increase in intensity, with extreme weakness and exhaustion, associated with a lethargic condition—resembling that of sleeping-sickness—and a brownish-red colouration of the portions of the mucous membrane which are opposite to the teeth (canine upper incisors, back-teeth). The tongue lies in the mouth motionless and without sensation, shrivelled and dry, dotted with ulcers, and sometimes gangrenous on the margins, from which a carrion-like odour is distinctly manifest. Palpation of the hinder part of the body, especially in the neighbourhood of the stomach, is very painful, and constipation usually continues till death. In rarer cases there exists from the first a sanguineous, uncontrollable diarrhœa. The palpebral conjunctiva is, like the mucous membrane of the mouth, coloured brownish-red, and the scleral conjunctiva is strongly injected, whilst purulent or mucous discharge from the eyes is always absent. The temperature does not rise at any period of the disease. With the appearance of somnolence and coma it is somewhat lowered; the cardiac impulse is strengthened and the pulse-rate quickened. There is no cough, the breathing is quiet and regular. The urinary secretion is diminished, and often suppressed (anuria), while the urine contains a copious quantity of albumen. Clonic convulsions are occasionally observed; also pneumonic symptoms (pneumonia of foreign bodies). Regarding diagnosis, the very sudden attack of vomiting and complete rejection of food, the somnolent and comatose condition, the striking vascular injection of the sclerotic, the browinsh-red colouration and ulceration of the mucous membrane of the mouth, are very characteristic symptoms. The course is acute; rarely hyper-acute. Death takes place, on an average, in from 4 to 6 days, with rapid emaciation and development of coma. In mild cases the disease lasts from 13 to 15 days; when the restoration to health is usually complete and no sequelæ are observed. The prognosis is unfavourable, the mortality representing 60 to 80 per cent. Young animals suffer less severely than older ones; a gradual development is also a favourable sign. The inception of the agent of infection (septic bacteria) apparently takes place from the digestive apparatus (primary gastritis and enteritis); the ulcers of the mouth appear to be secondary, and produced by the pressure of the teeth. The material of infection is of a fixed nature, and is conveyed either directly or indirectly by appropriate media. Medicinal treatment has proved useless. Alcohol aggravates the symptoms; only morphia, quinine and ichthargan with physiological salt-solution (administered by the rectum and subcutaneously) have sometimes been found to yield good results.

MALIGNANT CATARRHAL FEVER OF CATTLE.

Malignant catarrhal fever of cattle—Pseudo-malignant catarrhal fever of sheep—Malignant catarrhal fever of horses.

Etiology.—Malignant catarrhal fever is a disease which occurs only in cattle and buffaloes, and has nothing in common with human diphtheria. We possess no exact knowledge respecting its contagium. Its contagiousness, in comparison with that of other infective diseases, is very slight. Direct infection has been observed only in exceptional cases, and all the inoculation experiments which have been made, have given negative results. Its cause is probably a cowshed miasma, which becomes developed chiefly in damp, dirty sheds with defective drainage and bad ventilation. According to Frank, this disease is generally found on moderately moist, loose soil, and not on very wet or entirely dry subsoil. It is possible that the disease may also spread by means of intermediate bearers, as for instance, sheep (Möbius), or, as we have observed, cattle-dealers.

Malignant catarrhal fever is usually sporadic; although its appearance as an enzoötic throughout large cow-sheds and even villages is not rare. In some places it is a permanent enzoötic, and may break out during several successive years in the same shed. A great number of such cases are on record. Sump saw an instance of the disease remaining stationary for 25 years on a gentleman's estate, during which time 225 cows were attacked with the result of only 3 recoveries. Although young and well-nourished animals are most susceptible, many of mature age also suffer. It appears that the disease usually occurs in spring. We know nothing certain as to the influence of locality on this disease, which is found in high-lying places as well as in valleys and plains. The same remark applies to the influence of surface water. Bugnion and Frank state that the period of incubation is 3 or 4 weeks. Chill, which was formerly regarded as the original cause, has, as in other diseases, only a predisposing action. An animal may become repeatedly affected within a short period.

It has always been recognised that the disease assumes various forms, according to the respective localisation and intensity of the affection. Thus we speak of a nasal, abdominal, exanthematic, benign, and malignant form. The protean character of this disease in different animals has, for a long

time, given rise to inexact and confused ideas respecting its pathology.

Bacteriological Notes.—Nocard found, in the croupous membrane of the larynx and nasal fossæ, small ovoid bacteria, similar to those of hæmorrhagic septicæmia. Leclainche observed in the lymphatic system of the intestine and in the intestine itself, a variety of bacterium coli possessed of specific properties; the intravenous inoculation of which is asserted to have produced the development of catarrhal fever in a mild form. Bassi, however, regards catarrhal fever, not as an infectious disease, but as a poisoning by unwholesome hay (polytrincium trifolii).

Symptoms.—The symptoms of malignant catarrhal fever are exceedingly various and changeable. On the whole, this disease is a grave general affection, in which the mucous membranes of the eyes, respiratory organs, cavities of the head, alimentary canal, and urinary and sexual organs are attacked more or less severely, in a catarrhal, croupy or diphtheritic form; and there is severe nervous disturbance, especially of the functions of the brain. Usually, several organs or several functions are simultaneously affected, but occasionally only one suffers.

1. *Initial symptoms.* The disease generally begins rapidly with rigors and high fever, the temperature being between 104° and 108° F., even on the first or second day. The animal exhibits great torpor, supports its head on the manger, or holds it very low. The upper part of the head, especially the region of the frontal sinus and the base of the horns, is hot to the touch, and continued muscular tremor is noticed over the whole body. The hair is rough and stands on end, and the back is frequently arched. Even after the first few days, the patient sometimes becomes greatly emaciated.

2. *Eye symptoms.* Profuse lachrymation is generally the first well-marked symptom, in which case tears flow abundantly over the cheeks, and there is swelling of the eyelids, excessive congestion of the conjunctiva, and considerable photophobia, with consequent closure of the eyes. The swelling of the eyelids may lead to well-developed ectropium. In its further progress, this conjunctivitis becomes complicated with ordinary diffuse keratitis, in which case, the cornea becomes affected with an opacity which extends from the periphery inwards, and is at first dull and smoky, and later on milk-white. Ulcers on the cornea are rare. We may also have inflammation of the iris (iritis exsudativa and adhæsiva), in consequence of which there is an exudation into the anterior chamber of the eye, and adhesion of the iris to the capsule of the lens. Resolution may

take place; or the changes may lead to perforation of the cornea, complicated with prolapse of the iris, and atrophy of the eye-ball, which will be followed by permanent blindness. Persistent opacities of the cornea (leucoma) frequently occur. This affection of the eyes throughout its entire course closely resembles equine periodic ophthalmia.

3. *The respiratory symptoms* consist at first of catarrhal, and later on, of croupy and diphtheritic (sloughing) inflammation of the mucous membrane of the nose, larynx, bronchi, and accessory cavities of the head, with swelling of the lymph glands. In the early stage, the nasal mucous membrane is of a bright red or purple colour, but afterwards it becomes coated with a croupous and diphtheritic material which leaves ulcers behind. This inflammatory affection of the nasal mucous membrane is accompanied by a nasal discharge, which at first is sero-mucous, but soon becomes purulent, lumpy, mixed with streaks of blood, discoloured, and finally fœtid. Respiration is difficult, frequently stertorous, rattling, wheezing, and combined with mucous râles. The dyspnœa may turn into regular attacks of suffocation on account of the lumen of the larynx or nose becoming plugged up with croupy membranes. Croupy-diphtheritic masses are sometimes coughed up. When the inflammation spreads from the frontal sinus to the cavities of the horn-cores, inflammation of the matrix of the horn (which represents the periosteum of the processes of the horns) may ensue. The horns will then be very painful to the touch, and can be easily removed, or may even become shed.

4. *Digestive symptoms.* In severe attacks, the appetite decreases from the commencement, or is entirely suppressed. In slight cases, or when the disease is located in other organs, the appetite often remains good for a considerable time. During the first few days, the oral mucous membrane is considerably congested, dry, and hot. Later on, the animal becomes salivated from stomatitis, frequently to such an extent that the saliva flows in long strings to the ground. In such cases, the mucous membrane is infiltrated with hæmorrhages, and manifests, especially on the palate, cartilaginous pad of the upper jaw, and cheeks, erosions and insulated diphtheritic deposits, which vary in colour from yellow-grey [buff] to yellowish-white, and which leave ulcers behind after they are cast off. The necrotic inflammation may also spread to the skin of the muzzle and nose, in which case, the entire anterior part of the head may become swollen. Under these circumstances, the oral cavity

emits a very bad odour. Further on, we observe constipation, alternating with diarrhœa, and symptoms of uneasiness as if from colic. The fæces are fœtid, and resemble yeast or liquid manure. They are frequently mixed with blood, and not rarely contain membranous shreds. Frank states that croupy deposits from the intestinal mucous membrane of 6 to 9 feet in length are sometimes passed. Tenesmus is present at the same time, and the mucous membrane of the rectum is considerably swollen. The secretion of milk usually stops even during the first few days of the attack.

5. *Urinary and genital symptoms* are those of nephritis and cystitis. There is severe strangury. The urine often contains blood and the characteristic products of inflammation of the kidneys, such as urinary casts, epithelium of the kidneys, leucocytes, and albumen. According to Frank, its reaction is often acid. The vaginal mucous membrane is hyperæmic, swollen, and sometimes shows diphtheritic deposits, ulcers and a mucous discharge. Pregnant animals which are severely affected, frequently abort.

6. *Nervous symptoms* play the chief *rôle* in many cases of this disease, and consist, as a rule, in those of hyperæmia and inflammation of the brain. The animals become greatly excited and very restless, climb up on the rails, bellow loudly, shove up against their fellows, and keep to one side. They assume a wild, staring look, and frequently get into a regular frenzy ; or they may exhibit attacks of extremely varied kinds of spasms, such as convulsions, epileptiform fits, rolling of the eyes, and even seizures resembling trismus. The spasms often change later on into paralysis, or merge into a condition of general weakness and increasing coma, in which case the disease ends in death, preceded by a fall in the internal temperature.

7. *The skin* becomes occasionally affected, in which case we find an eruption of vesicles or nodules, falling off of the hair, and, subsequently, severe desquamation of the epidermis, for example, on the udder, inner side of the joints, interdigital space, inner surface of the thighs, throat, and back. The hoofs may become shed in rare instances. Strebel states that a bilateral inflammation of the hock has sometimes been observed.

Anatomical Conditions.—We find as much variety in the anatomical changes as in the symptoms, according to the duration, localisation, and severity of the disease. Slaughtered animals show chiefly initial changes, such as catarrhal or croupy

processes; and those which have died from the disease, true diphtheritis of the mucous membranes. The following are the most important points that have to be considered in the examination of the respective organs, which are by no means simultaneously affected in all cases.

The mucous membrane of the *nasal cavity* is of a brownish-red or purple colour; and is swollen, infiltrated with hæmorrhages, covered with whity-yellow, croupy masses, and is ulcerated. The sloughing process sometimes spreads to the turbinate bones and ethmoidal cells. The mucous membrane in the frontal sinus is also swollen and covered with pus; there is an accumulation of pus in the cavities of the horn-cores; the fleshy walls of the horns are inflamed; the mucous membrane of the larynx and bronchi shows simple catarrhal, or hæmorrhagic and croupy inflammation; the croupy coating often extends into the small bronchi; and the lungs very frequently exhibit interstitial emphysema, and also œdema in fatal cases.

The mucous membrane of the *oral cavity* is of a purple colour and swollen, particularly near the teeth. It is said that vesicles, which rapidly change into superficial erosions, may be seen on it. These erosions, which are not rare, may be larger than a sixpence. In severe cases, the mucous membrane, especially of the soft and hard palate, becomes covered with insular whitish-yellow membranes, which frequently cover the greater part of the pharyngeal cavity and may extend even into the œsophagus. The mucous membrane of the pharynx generally shows the same changes as those of the oral cavity. The mucous membrane of the stomach and intestines is congested diffusely and in spots, swollen, and often covered with diphtheritic deposits and ulcers, which may leave scars after healing. Peyer's patches and the solitary follicles are usually swollen. Thus, the aspect of the disease in many cases may closely resemble that of rinderpest.

The *cavities of the skull* are rich in blood, especially in the cerebral meninges; and there are slight hæmorrhages and œdema in the brain and its membranes. Inflammation of the brain has also been observed. We find similar changes in the spinal cord. The kidneys are inflamed and show fatty degeneration. In the pelvis of the kidney we sometimes find croupy deposits which may extend to the ureters and bladder. The mucous membrane of the bladder is inflamed in extremely varied degrees, is often covered with croupy membranes, and infiltrated with hæmorrhages. Among general changes we may observe: hæmorrhages throughout the entire body, especially in the

heart, under the serous membranes, in the mesentery and omentum, and under the skin; fatty degeneration and swelling of the liver; fatty degeneration and discoloration of the muscles; rapid putrefaction of the cadaver; dark-coloured and thick blood; accumulation of sero-sanious fluid in the cavities of the body; slight inflammation of the pleuræ; etc.

Course and Prognosis.—The course of malignant catarrhal fever is sometimes acute and even peracute; at other times subacute, and even chronic. Death may take place as early as from 3 to 5 days after the attack, although recovery may be delayed for several weeks. The disease lasts on an average 3 or 4 weeks. Relapses are so frequent that an acute seizure may take place as late as the second or third week.

During the first few days, we may often observe an apparent improvement, which may be quickly followed by a severe exacerbation. The period of convalescence is always more or less protracted. In mild cases, complete recovery takes place in about a month; but may be retarded for double or treble that period in severe attacks, except when the course of the disease has a rapidly fatal termination.

Prognosis is always very doubtful. The mortality varies from 50 to 90 per cent. Among 76 cases, observed by Bugnion, only 7 recovered. All the others were slaughtered on the third or fourth day after they became ill. Frank saw only 6 per cent. of the treated animals recover. Unfavourable factors are: simultaneous extension of the disease to several organs, or to all the organs of the body; development of the inflammation into necrosis of the affected mucous membranes; and grave cerebral, pulmonary and intestinal attacks. Besides the previously described changes in the eyes, we have, as a sequela, the development of chronic catarrh of the sinuses of the head.

Differential Diagnosis.—Most important of all is the differentiation of malignant catarrhal fever from rinderpest, which is by no means easy on all occasions. The following are points to be considered: 1. The contagiousness of malignant catarrhal fever is as slight as that of rinderpest is great. 2. In malignant catarrhal fever, the best-marked symptoms are those of the eyes and respiratory organs (stridor and nasal discharge); and in rinderpest, those of gastric disturbance. The same

remark applies to the *post-mortem* appearances of the respective diseases. 3. The course of malignant catarrhal fever is, as a rule, not so acute as that of rinderpest. The differentiation becomes much more difficult when several animals are affected with malignant catarrhal fever in the same shed, and when the intestinal canal is specially affected with simultaneous implication of the vaginal mucous membrane. Here we may try inoculat on, the negative result of which will be a certain sign of catarrhal fever. This disease may also be mistaken for meningitis, dysentery, periodic inflammation of the eyes, and benign rhinitis fibrinosa (Berndt), in which we find neither opacity of the cornea nor any nervous symptom. This rhinitis is, however, infectious, and sometimes appears as an epizoötic.

Therapeutics.—Frank, Hink and others have recently called attention to the importance of thorough disinfection and draining of the subsoil in those cow-sheds in which the disease is stationary. It has also been recommended to render the floor of the shed waterproof. The infective nasal discharge should be frequently removed and thoroughly disinfected. Segregation is also advised. No remedy has as yet been found. In former times, blood-letting, washing the skin with vinegar, inhalations of vinegar, sawing off the horns, trepanning, and the administration of tartar emetic and camphor were recommended. Recently, many authorities, such as Frank, Hink and Zündel, say that they have obtained successful results with the internal administration of creolin and carbolic acid (1¼ to 2½ drams), and with external applications of these agents in the form of washes, inhalations, and ointments. Hink strongly recommends washing out the nasal cavity with a 2 per cent. solution of creolin. When severe cerebral symptoms are present, we recommend the continued application of cold to the head, as for instance, by means of bags of ice and cold douches. This treatment has been in vogue for a long time. The other symptoms should be treated symptomatically with antipyretics, alkalies, expectorants, emollients, etc.

Pseudo-malignant Catarrhal Fever of Sheep is a contagious infective disease which possesses an extraordinary likeness to distemper of dogs, and which is characterised by an affection of all the mucous membranes, especially those of the nasal cavity, eyes, and respiratory organs. The investigations of Friedberger point to the probability that the malignant catarrhal fever of sheep is caused by a contagium which may remain in a shed for several years, and which can evidently be transmitted to a certain distance; because animals which were in

healthy and separate sheds have become infected. Audum believes that he has found the contagium in the form of a bacillus of from 1 to 3 μ long, and of the thickness of a tubercle bacillus. The disease usually occurs enzoötically, and in some instances, epizoötically. It is with justice greatly feared. Formerly it was thought that this disease was caused by simple coryza becoming "malignant," "degenerating," or passing into "glanders." Other authors speak of an epizoötic form of benign catarrhal fever. This disease is not identical with the catarrhal fever of cattle. According to Theiler, the disease also occurs in South Africa (the blamvtong).

The disease begins with symptoms of suppurating nasal catarrh. A ropy mucous discharge collects round the nostrils, which often become glued together, and from which a muco-purulent and (according to some writers), sometimes, a fœtid substance can be squeezed. The nasal mucous membrane is highly congested and swollen, and symptoms of laryngitis (coughing), bronchitis, and in severe cases even symptoms of bronchial pneumonia or pneumonia caused by foreign bodies may appear at the same time. Suppurating blepharitis, purulent conjunctivitis, keratitis, and even ulcers and abscesses of the cornea are also seen. The eyelids are reddened, swollen, and glued together by a greasy, purulent substance. The eyelashes below the inner canthus are wet and soiled with a yellowish-brown greasy discharge. The conjunctiva varies in colour from orange to vermilion-red, is swollen, and often secretes enormous masses of purulent secretion (blennorrhœa). The skin near the orifice of the sheath suffers from a suppurating dermatitis, accompanied by catarrh of the prepuce. We find symptoms of intestinal catarrh, such as constipation, diarrhœa, straining to defecate, and suppressed appetite. The general health is greatly upset; and the animal becomes depressed and anæmic, rapidly loses condition, is slightly feverish, and staggers and reels when walking. Finally, the patient, being too weak to stand, falls down and lies on the floor in deep sleep, which sometimes is interrupted by symptoms of approaching death, such as clonic spasms of the muscles of the limbs and trunk, grinding of the teeth, etc. The course of the disease is somewhat acute, and prognosis is very unfavourable, especially in lambs.

On *post-mortem* examination we find the mucous membrane of the nasal cavity, particularly its upper parts, to be congested in a more or less diffuse and ramiform manner, especially the mucous membrane of the turbinate bones, which varies in colour from scarlet to very dark red or chocolate, and is swollen, very shiny, and covered with a purulent secretion, which in some parts may look like frog-spawn, and in others may be creamy, lumpy, caseous, and of a dirty dull-yellow colour. Erosions and ulcers are sometimes found after the removal of this coating. In the other air-passages the changes are those of catarrhal bronchitis, inflammatory œdema of the lungs, broncho-pneumonia, and pneumonia caused by foreign bodies. The liver is pulpy, brittle, infiltrated with fat, and of a clay colour. The epithelium of the kidneys shows a high degree of fatty degeneration; the brain is slightly œdematous; the muscles of the heart are relaxed, brittle, and of a dull-red colour; the blood is very thin or manifests no tendency to coagulate; the muscles of the body are pale; the cadaver is greatly emaciated; and ecchymoses are found in various organs. *Prophylaxis* consists in

the respective separation of the healthy and of those which are but slightly affected; in thorough disinfection of the sheds; and in the supply of good nourishing food, such as mashes and bruised oats. Tonics, such as sulphate of iron, and bitters in the form of electuaries may be given in mild cases. It is best to slaughter those animals which are severely affected; for they are generally beyond hope of recovery.

Malignant Catarrhal Fever of Horses (?).—During an outbreak of catarrhal fever in cattle, Hable saw two horses which were affected with a disease that very closely resembled it. One of these horses had a purulent discharge from the nose and mouth, opacity of the cornea, swelling of the eyelids, lachrymation, difficulty in swallowing, considerable swelling of the pharyngeal lymph glands, swelling of the head, and inflammation of the lungs. This animal died on the fourth day of the attack. The other horse presented similar symptoms; the opacity of the cornea and swelling of the eyelids being particularly well-marked. He recovered, but remained blind in consequence of opacity of the cornea.

ENZOOTIC ABORTION.

Etiology.—Abortion occurs as an infective disease among cattle, and less frequently among sheep, goats, pigs, and horses, at certain times, and in certain stalls, pastures, and districts. On some estates it appears every year. It occurs enzoötically and sometimes even epizoötically; whilst ordinary abortion is observed only sporadically, in consequence of mechanical injuries, acute feverish attacks, chill, poisoning, and partaking of food and drink which contain fungi and other noxious material. The infective matter differs in horses and cattle.

1. According to Bang and Stribolt, the abortion of cows is produced by a small bacillus (bacillus of abortion). As in the case of the human gonococcus, it passes through the vagina and cervical mucous into the uterus, and there excites a specific uterine catarrh, which leads to the death and expulsion of the fœtus, when the abortion bacillus has passed through the amniotic fluid to the latter. Inoculation experiments with pure cultures of the abortion bacillus, by injection into the vagina, produced abortion in pregnant cows and ewes; the same result has followed intravenous injection in ewes and mares. The vitality of the bacillus of abortion is very great. After the consequent abortion, the bacilli remain active in the uterus of the infected cow for many months, and may even lead to the occurrence of abortion in a subsequent pregnancy. Bulls play an important part as agents in the transmission of the infection;

for they retain the abortion-bacillus for a long time in the prepuce in a vigorous condition, without displaying any symptoms of disease, and must be regarded as the chief distributors of this malady. Frequently, also, a direct inoculation of a healthy neighbouring cow takes place through the uterine secretion, the amniotic fluid, the placenta, or the dead fœtus of the affected animal. The disorder is often introduced by the buying of cows from polluted stalls, by straw, the manure of the byres, and by attendants and veterinary surgeons (as in the removal of the placenta without subsequent disinfection). Its extreme contagiousness has already been frequently shown by the clinical experiment of artificial transference of the vaginal mucus of a cow, which had aborted, to that of a healthy cow, and the subsequent abortion which, as a rule, took place after from 9 to 21 days (Brauer, Lehnart, Trinchera, etc.). In many cows a single abortion suffices to confer immunity, either transitory or permanent. In others the tendency to abortion ceases after the second or third time of its occurrence. As a rule, abortion spontaneously ceases in an affected farm-yard after some 2 to 4 years, if fresh cows are not being constantly introduced (Sand, Bang).

According to Bang and Stribolt, the bacillus of abortion of the cow is found in the yellow, inodorous exudate which is situated between the membranes of the embryo and the uterine mucous membrane, in the form of a pure culture. The bacilli are small, and of unequal length ; the larger being somewhat of the size of a tubercle-bacillus, and the smaller resembling cocci ; they have 1 to 3 nuclei ; stain with the ordinary aniline dyes, but not with the Gram re-agent ; and possess no spontaneous movement. The cultures thrive best on a mixture of gelatine, agar, and fresh serum ; where they grow only in a circumscribed zone of 0·4–0·6 inches in thickness and reaching to about 0.2 inch beneath the surface. The colonies are very small, punctiform, roundish, the largest being hardly of the size of a pin's head. These bacilli grow best—on the one hand, with a low, on the other with a high oxygen tension. In contrast to this, the ordinary proportion of oxygen present in atmospheric air (21 per cent.) is obstructive to their growth, as is also a complete absence of oxygen. According to Preisz, the bacillus of abortion (corynebacterium abortus endemici), when placed in a warm room will not multiply, and forms no spores ; while on the other hand, it propagates anærobically, on the chilled blood-serum of the calf, and also on peptonised broth.

2. The researches of Ostertag (Graditz, Hoppegarten) show that the enzoötic abortion of mares is produced by streptococci (the streptococci of abortion), which may pass into the pregnant uterus either from the vagina or with the blood-stream; and there set up a catarrh of the uterine mucous membrane, which leads to a loosing of the natural connexion between the uterus and the membranes of the embryo, and thereby to a premature expulsion of the fruit. The mares, which have taken in the cocci of abortion, protect them, and, after abortion has taken place, discharge them from the genital organs, and can thus give rise to the distribution of enzoötic abortion. The infective agents in the abortion of foals and that of calves are not identical, as Bang conjectured. Ostertag failed to produce abortion of calves, either by the introduction of pieces of the membranes or portions of the amniotic fluids of aborted foals, or by inoculation of the cocci of abortion on pregnant cows. He has also demonstrated that there is no etiological relationship between enzoötic abortion and the paralysis of foals.

Symptoms.—This form of abortion occurs in cows usually during the third or seventh month of pregnancy. The premonitory symptoms are: swelling of the udder; great congestion of the vaginal mucous membrane; dirty-red (whity in mares), catarrhal, muco-purulent discharge from the vagina; occasional formation of pimples the size of a millet-seed on the mucous membrane; decreased yield of milk; and a colostrum-like condition of the milk. Abortion generally occurs about 3 days after the appearance of the discharge, with strikingly mild general symptoms. The fœtus, as a rule, is born dead. A persistent vaginal discharge often remains for a considerable time after the abortion, in consequence of which the cow may suffer from continued bad health, and may become sterile.

In mares, abortion usually occurs in the fourth or ninth month of pregnancy. Guillerey distinguishes a mild and a malignant variety. The first is the more frequent, and occurs from the fourth to the seventh month of pregnancy, with an incubation stage of 12 days; and usually progresses to recovery in a period of from 4 to 5 days. The malignant form has an incubation of 4 days, and appears from the seventh month onwards, being characterised by difficult labour pains of extreme severity, arthritis of the tarsal and knee joints, tendo-vaginitis, foundering, hæmoglobinuria, phlebitis, mastitis and other complications. According to Konge, in cases of infective abortion

in contrast to the traumatic ones, a rise of temperature up to 103° to 104° F. appears always, as a diagnostic symptom, on the second and third days after foaling.

Therapeutics.—1. Prophylaxis is the chief thing to be attended to in cases of enzoötic abortion. If the act of abortion once begins, it cannot be stopped by the administration of opium or any other remedy. The most important and frequently the only effective measure is to transfer the pregnant cows to another shed as soon as possible; or, if practicable, to turn them out to grass and to rigidly isolate them as regards feeding and tending. It is also imperative to completely remove and to render innocuous every after-birth and dead fœtus. Each stall and the entire stable should be thoroughly disinfected; * the patients should be disinfected by washing the genital organs with a watery solution of creolin, lysol, or carbolic acid, and injecting a similar solution (½ to 2 per cent.) into the vagina and uterus. Reindl recommends a carbolised soda-solution. Cows which have aborted should not be brought into their old stalls, or into contact with the healthy cows, till after a prolonged interval, and when they are again perfectly healthy. Affected animals, too, should never be encouraged to breed again till the vaginal discharge has completely ceased. It is advisable to disinfect the prepuce and penis of the bull after every covering of a suspected cow; which procedure should also be applied to a bull that has been just purchased. The fact that the change of a bull has sometimes a good effect on this enzoötic disease, may be explained by the exclusion of the former ones as bearers of infection. As newly-purchased animals, especially, may prove to be media of infection, they should be kept in a separate place at first and carefully examined. The sale of cows which have aborted and their replacement by new purchases is, according to Bang, an unsatisfactory means of stopping the disease as the newly-arrived cow may, in many instances, become infected in the unhealthy stall. Grazing sows which might infect themselves by the vaginal discharge of affected animals, and those which have aborted, should be kept away from the pastures for a few weeks (Gassner). In order to decrease the predisposition to the disease, we might try to strengthen the constitution of the animals by exercise and nitrogenous food.

Finally, Bräuer attempted to protect the animals against

* Bang advises the use of quicklime; as, besides being efficient, it neither taints the milk, nor diminishes the value of the manure.—Tr.

the disease by means of subcutaneous carbolic injections (two or three Pravaz syringefuls of a 2 per cent. solution) under the skin of the flanks, every fortnight, from the fifth to the seventh month of pregnancy. He states that he obtained good results with these injections in a series of cases. Some of the recent reports on the efficiency of this method are positive; others negative (Schleg, Bucher, Wilhelm, Kuhn, etc.). It is probable that the alleged favourable results were due to the simultaneous disinfection of the external genital organs; as well as to the fact that abortion in case of infected cattle usually ceases to occur after a period of from 1 to 3 years, as a result of natural immunisation of the cows.

2. In the case of mares, the most important considerations are, according to Ostertag, the combating of the enzoötic abortion, and the innocuous removal of the products of abortion with the embryonic membranes. There must also be a thorough disinfection of the stall and of the discharges with sublimate solution (1 : 1000). The infected pasture should not be grazed on by pregnant mares for at least three months, and the attendants should disinfect their hands and boots. The uterus of the affected mare, from immediately after the occurrence of the abortion to the closure of the os and the cessation of the vaginal discharge, should be irrigated twice a day with a lukewarm, ½ per cent. lysol solution; and the neighbourhood of the vaginal orifice should be disinfected at the same time. The mares should not again be covered before the lapse of six weeks after the completion of the normal period of pregnancy. The penis and prepuce of a stallion that has covered a mare after a preceding abortion should be thoroughly disinfected after the act of copulation with a lukewarm ½ per cent. lysol solution; and veterinary inspectors should insist that all stallions be thus treated.

DYSENTERY.

General remarks—Dysentery of sucklings—Dysentery of man—Dysentery of adult animals—Bovine dysentery from coccidia.

General Remarks.—The recent investigations of Jensen on the dysentery of sucking calves, and those of Zschokke, Hess, and Guillebeau on that of full-grown cattle, have thrown some light on the nature of dysentery in our domestic animals. Dysentery in them may be divided into dy-

sentery of sucklings and dysentery of full-grown animals. These two forms must be regarded as independent diseases. The dysentery of horses, dogs, sheep, and birds has, however, been but little studied. The question whether the dysentery of our domestic animals is etiologically identical with that of man, still remains unanswered.

The Dysentery of Sucklings.

Occurrence.—The dysentery of sucklings chiefly occurs in calves; though it also affects pigs, lambs, foals, cats and dogs. Next to pyæmic polyarthritis, it is the most destructive disease peculiar to sucklings. For several years it has steadily increased in various provinces in Germany. Many farms have been so severely attacked, that the raising of young cattle has been rendered impossible. Röll reports that, out of a total of 3,318 calves, 1,196 became infected, and 1,152 died (a mortality of 97 per cent.). In the year 1884, 55 per cent. of all the affected animals died in some parts of Austria. It attacks calves almost exclusively during the first 3 days of life. The animals often become affected directly after birth, even before they have taken any milk, a fact which goes far to prove that the mother's milk does not participate in the production of the disease. A further proof of the non-participation of the mother's milk in the transmission of the disease, is the practical observation, that dysentery also appears when milk from non-affected sheds or boiled milk is given, and even when substitutes for milk are employed.

Etiology.—The cause of the dysentery of calves is, according to Jensen, an oval bacterium which appears singly, in pairs, or in the form of chains, and is either a pathogenic variety of the bacterium coli commune or identical with it. Jensen considers that the disease is not due to a specific contagium, but to a pathogenic variety of the bacterium coli commune, which is normally present in the intestine. The usually harmless bacterium coli commune may become pathogenic, and may penetrate, as the exciter of the disease, into the intestinal mucous membrane, when the newly-born calf becomes exposed to debilitating influences, such as chill, errors of diet, feeding at first with boiled milk, etc. On the first day the resistance to the bacterium coli is but slight; it afterwards increases with the onset of the normal digestive processes (glandular secretions),

and as the system becomes accustomed to the presence of bacteria. Cultures of the intestinal bacteria, which have become pathogenic, also produce dysentery in calves previously quite healthy. The intra-peritoneal inoculation of guinea-pigs is productive of a fatal, sero-fibrinous peritonitis which runs a very acute course. Schütz has confirmed the results of Jensen's bacteriological researches. According to him, the intestine is the source of the infection. The contagium is disseminated throughout the shed by the fæces of the infected calves, and gets into the bodies of healthy calves by means of intermediate bearers, such as farm servants, and the dung-soiled teats of cows. According to Joest, an infection per os in calves, suffering from inactive digestive organs, is more readily produced than with older animals. The observations of Nocard further show that in the transmission of dysentery, an infection per os is not absolutely necessary, and that infection may come from the umbilical cord, in calves, twelve hours only after birth. Intravenous injection of the smallest quantity of the bacillus coli will kill calves very quickly under the most serious signs of general infection. Dysentery in calves is therefore, according to Joest, not to be regarded as enteritis, but as septicæmia.

According to the more recent researches of Poels and Nocard, the term "calves' dysentery" indicates no single, specific, infective disease; but constitutes a group-name for a number of diseases which are etiologically distinct. Calves' dysentery is not exclusively a colibacillosis—that is to say, an enzoötic disorder produced by colon bacilli—but it can also be produced by the bacteria of hæmorrhagic septicæmia, streptococci, bacillus pyocyaneus, the pseudo-coli-bacillus, and other micro-organisms. According to Nocard, the dysentery of calves which appeared in Ireland, where it was known as "white scour," is identical with the infectious "lung disease" which prevailed at the same time in that country; and is produced by a very small, non-motile bacterium, which is not stained by the Gram re-agent, and belongs to the Pasteurella group (Pasteurellosis).

Anatomical Conditions.—There is nothing specially characteristic about the anatomical changes. The intestinal mucous membrane is covered with mucous and pus, which contain a great quantity of bacteria. The epithelium of the mucous membrane is absent in some places; and the mucous membrane itself is loosened and swollen, and sometimes shows a superficial diphtheria of Peyer's patches. In very severe cases, the contents

of the intestines are more or less bloody (hæmorrhagic intestinal inflammation). Ulceration does not occur, apparently on account of the short duration of the disease. The mucous membrane of the abomasum, especially on the top of the folds and in the region of the pylorus, is hyperæmic; it contains ecchymoses, is œdematously swollen, and is frequently in a state of maceration. The contents of the abomasum generally consist of caseous coagula, and has a normally acid reaction. We have sometimes found in it traces of blood. The excessive general anæmia of the cadaver, especially of the internal organs, such as the liver and the kidneys, and the parenchymatous changes of the liver, muscular system, and heart, are very striking. A lobular broncho-pneumonia may also develop. In England, after the occurrence of calves' dysentery, the appearance of pulmonary inflammation, with formation of cavities, constitutes a very frequent complication.

Symptoms.—The symptoms of the dysentery of sucklings are exactly the same in the different species of animals.

The *calf*, during the first few days after birth, loses more or less its desire to suck; it suffers from pappy diarrhœa and tenesmus, is restless and lows. Later on, the excrements, which at first were yellow, become whitish and very thin; they assume a mucilaginous condition, are mixed with coagulated milk (so-called "white scour") and frequently with blood, and are then very fœtid. Finally, the fæces are passed involuntarily; there is excessive weakness; and the animal lies down continuously, suffers from convulsions, and allows the saliva to flow from the mouth. Death often takes place within 24 hours, although the average period of the duration of the disease is from 1 to 3 days. The mortality varies from 80 to 100 per cent. Very frequently all the calves of a shed die one after the other. The survivors generally remain sickly for a very long time (chronic pulmonary inflammation, arthritis, stomatitis).

Lambs, also, give up sucking; they become depressed and very weak; and, suffering from diarrhœa accompanied by tenesmus, they pass thin, mucilaginous, fœtid fæces. At first the temperature may rise to 106° F., but rapidly falls towards the end (Nikolski); respiration is accelerated, and saliva and mucus flow from the mouth. As with calves, the duration of the disease varies from 1 to 3 days, but death sometimes ensues in a few hours. The disease in lambs is also specially

prevalent during the first 3 days of life. According to an observation reported by Nikolski, 30 per cent. of the infected lambs become ill on the first day after birth, 40 per cent. on the second, 25 per cent. on the third, and only 5 per cent. subsequently. He also mentions that, out of a herd of 12,000 sheep, 50 per cent. of the lambs died from this disease.

In *foals* dysentery is also found during the first 3 days after birth (Mazoux), and sometimes later on. The animal becomes depressed and restless ; the frequently-passed fæces are fœtid, or thin and mucilaginous ; the breath and the exhalations from the skin often have a very bad smell ; the eyes are sunken ; the animal becomes very weak, very thirsty, and greatly tucked up in the flanks ; and sometimes there is a cutaneous eruption, which spreads over the whole body, and is particularly well-marked about the anus.

Diagnosis.—We diagnose dysentery from the fact of its enzoötic character, and from the presence of severe and rapidly fatal diarrhœa during the first few days of life. In sucklings, the disease is most readily mistaken for acute simple gastro-intestinal catarrh, due to errors of diet and defective condition of the milk of the mother. This latter affection of the alimentary canal does not, however, as a rule, become developed immediately after birth. Its course, further, is milder than that of dysentery, and the fæces present a different colour—a loamy-yellow to a greyish-green. On the part of the farmers, too, the epizoötic so-called "calves' death," and also cases of pyæmic umbilical inflammation were usually confounded with this condition. (See chapter on Pyæmic Polyarthritis.)

Therapeutics.—The sick animals should be segregated, the infected sheds thoroughly disinfected, the udder most carefully purified, and the female organs washed out with a disinfecting fluid, both before and after parturition. If the disease is enzoötic in a shed, pregnant cows should be put a few days before calving into a fresh and clean shed. The observance of these precautions has proved to be far more effective than all the medicines that have been used against dysentery. As "calves' dysentery" may also arise from infection of the umbilicus, a thorough disinfection of that part should be carried out immediately after birth.

It is advisable at the first appearance of the disease to give a mild laxative, such as a few spoonfuls of castor oil for calves

and lambs, and small doses of calomel (1½ to 3 grains *pro dosi*) for foals. Usually, rhubarb root (30 to 75 grains for calves, and 7½ to 15 grains for lambs) and opium (15 to 30 grains for calves, and 1½ to 3 grains for lambs) are given alone or combined. We can recommend the following mixture: pulv. rhei radix 75 grains; pulv. opii 30 grains; and pulv. magnes. carbon. 15 grains; to be given in one dose to a calf with 3½ oz. of camomile tea, or 1½ oz. of whisky. Instead of opium, we may give its tincture, 1¼ to 2½ drams for calves and foals, and 15 to 30 minims for lambs. Opium and its tincture should be given in much larger doses than were formerly employed. The doses mentioned may be repeated (on an average 3 times with intervals of 6 hours) until the diarrhœa has ceased. Also tannin (15 to 30 grains for calves, and 3 to 7½ grains for lambs) may be given either alone or combined with an equal quantity of salicylic acid, as for instance, 30 grains of each in camomile tea, once or twice a day for calves. This combination of salicylic acid and tannin is greatly esteemed by many persons. Also tannalbin (45 to 75 grains for a calf), tannopin (1 to 2 drs.), tannoform (7½ to 15 grains), nitrate of silver (1 tablespoonful of a ½ per cent. watery solution every 3 hours for calves), creolin and lysol (three doses daily of 15 minims in water for calves), tar-water (doses of ½ pint for calves), resorcin, naphthalin, salol, salts of bismuth, argentum, colloidale, ichthargan, and other remedies have been recommended. We may use with advantage mucilaginous demulcents, such as linseed tea, mucilage, decoction of althæa, oatmeal gruel, raw eggs, lime-water, either alone or combined with opium.

Serum inoculations have recently been recommended by Jensen in the treatment of "calves' dysentery." As a single definite coli-serum is, however, not protective against any variety but its own, a polyvalent serum must here be employed; just as in the treatment of swine-fever (Joest). The serum-inoculations carried out in the province of Saxony would appear to have been effective (Raebiger).

Prophylactic Precautions against Calves' Dysentery according to Poels.—In Holland very good results have been obtained from the following procedures recommended by Poels for the prevention of "calves' dysentery":

(*a*) *Observances before calving.* 1. The tail of the cow is tied up with a string, by which the tuft is secured to a rope placed round the neck, so that the tail rests against the flank. 2. Before the discharge of the waters,

the hinder parts of the cow, especially the vulva and the udder, are carefully cleansed with a sponge dipped in a 3 per cent. creolin solution (10 tablespoonfuls of creolin to a gallon of water). 3. The vagina is rinsed out with a solution of 15 grains of sublimate in rather more than a gallon of water, and applied with a specially prepared syringe. 4. The vicinity of the vulva is cleansed with a special brush and a sublimate solution. 5. All excrements are removed; and all soiled parts of the body cleansed with a creolin solution. 6. Care is taken that neither the membranes nor the calf become soiled with the intestinal evacuations.

(*b*) *Observances during and after calving.* 7. The cow is bedded on clean straw, and the calf is caught on a clean linen cloth. 8. The navel-string is tied as closely as possible to the trunk with a cord previously boiled, or dipped into one of the solutions, Nos. 3 and 4, named above, and is then cut off close to the seat of the ligature. 9. The stump of the navel-string is then washed with a solution of 75 grains of permanganate of potash in a quart of water. 10. The calf is cleansed of adhering mucus, especially at the mouth and nose. 11. A closely-woven muzzle is placed on the calf, which, during the first six or seven days, is removed only at the time of drinking. 12. The calf is brought into a clean, quiet shed, free from draughts, and fairly warm. 13. A little of the first milk is drawn off from each of the cows' teats; and some two quarts is then milked into a clean vessel. This is given to the calf, and the process is repeated after half an (or a whole) hour. 14. The milk-supply of the calf, which should always be supplied from its own dam, is, for the first 24 hours, 3½ to 4½ quarts; on the second day, up to 6 to 7; on the third, to 10 to 11; on the fourth, to 12 to 13; on the fifth, to 12 to 13; and on the sixth day, to 17 to 18 quarts.

The Dysentery of Man is essentially an infective, croupy, diphtheritic inflammation of the large intestine, with suppurating and hæmorrhagic infiltration of its mucosa and submucosa. Its virus, the nature of which is unknown, is probably not directly contagious. The infection is communicated by the evacuations of persons suffering from dysentery. The disease appears sporadically and endemically, and in our climate is on the whole benign, but is very malignant in the tropics. The symptoms consist of gradually increasing diarrhœa and tenesmus; colic; vomiting; mucous and subsequently purulent, and even bloody condition of the fæces; pains during micturition, and grave general disturbance. Complications sometimes set in, as for instance, abscess of the liver, a diseased condition of the articulations, inflammation of the serous membranes, perforative peritonitis, etc. Therapeutics consist chiefly in the administration of aperients (calomel and castor oil) and stimulants. [The most valuable drug in the treatment of human dysentery is undoubtedly ipecacuanha.—TR.]

THE DYSENTERY OF ADULT ANIMALS.

Occurrence.—This disease seems to occur most frequently in cattle, and is more common in stall-fed animals than in those on grass. It often ceases when the animals are turned out on

pasture. It may sometimes remain for several years in a shed, in which case it seems to gradually assume a milder character. It may, however, break out sporadically. Such factors as chill, errors of diet, over-exertion, damaged fodder and bad water, bad seasons and times of war, which were formerly supposed to be its causes, have only a predisposing influence.

Anatomical Conditions.—The mucous membrane of the large intestine is more or less hyperæmic and hæmorrhagic. It is often swollen; in parts denuded of epithelium; and covered with yellow diphtheritic deposits, which, later on, leave ulcers and scars. The contents of the intestine are fœtid, and their colour varies from dirty-yellow to that of blood. The mucous membrane of the abomasum is also hæmorrhagically swollen, and the intestinal lymphatic glands, especially Peyer's patches, are inflammatorily infiltrated and ulcerated. In chronic cases, the intestinal mucous membrane is sometimes thickened and of a slate colour; the muscular coat of the intestine, hypertrophied; and the intestinal serosa in the neighbourhood of the ulcers and scars, affected with adhesive inflammation.

Symptoms.—As a rule, the disease manifests itself suddenly by loss of appetite, high fever (104° to 106° F.), rigors, debility, attacks of colic, and diarrhœa. Later on, the fæces become very thin, mucilaginous, frothy, discoloured, fœtid, and even bloody. They are often mixed with undigested food, croupy membranes, and cast-off epithelium from the mucous membranes. At the commencement of the attack, tenesmus, and frequent, unsuccessful and severe straining to defecate are present, so that prolapse of the rectum may take place. Later on, however, the fæces flow involuntarily from the paralysed, gaping anus. The mucous membrane of the rectum is excessively congested and swollen. The animals evince pain on palpation of the hind-quarters. On pressing the right flank with the hand, we may sometimes feel well-marked fluctuation of the intestines, which are filled with fluid (Albrecht). Weakness and emaciation increase; the internal temperature falls below normal; and death often ensues in as short a period as 1 or 2 days, but usually not until 2 to 4 days. Mild cases recover in 2 or 3 weeks. The chronic course of cases which continue for weeks or months is probably due to the presence of intestinal ulcers. The prognosis, even in grown-up animals, is always doubtful. The mortality is about 50 per cent.

Differential Diagnosis.—In full-grown cattle, it is difficult, and may be impossible, to differentiate dysentery from toxic, mycotic, and enzoötic intestinal inflammation. The results of *post-mortem* examination of cases of dysentery sometimes closely resemble those of rinderpest. It is important to remember that in dysentery, only the intestinal canal is affected, and that a direct infection can never be proved. A distinction between dysentery due to a toxic or mycotic inflammation of the intestines is not always possible.

Treatment.—Besides mucilaginous agents, such as linseed tea and a decoction of althæa, we give opium (2½ to 6½ drs.) and astringents, such as sulphate of iron, tannic acid, sugar of lead, and nitrate of silver. Coarse fodder should be avoided.

Bovine Dysentery from Coccidia.—Zschokke, Hess, and Guillebeau have closely studied a form of dysentery which occurs among cattle in Switzerland, and to which they have given the names of red dysentery and dysenteria hæmorrhagica coccidiosa. It attacks young animals, especially those on grass, enzoötically; and older ones, sporadically. On a pasture, or in a cowshed, it spreads from one animal to another in rapid succession, until several or all of the cattle become affected. It appears chiefly when the animals are fed on grass, and when they are turned out to pasture, and consequently in summer and autumn. It is supposed that the coccidia, which are specific infective protozoa, are received into the system along with the food and water, and that they immigrate into the epithelium of the intestinal mucous membrane, and set up severe inflammation in it. The disease can be produced experimentally in cattle by sporulating coccidia.

The period of incubation is about 3 weeks. In slight cases, the general health is at first but little disturbed. In severe cases there are rigors, and the temperature rises within the first 24 hours to 105·8° F. In from 2 to 6 days, the animal evinces great weakness of the hind-quarters, has difficulty in getting up, and persistently lies down; the upper eyelids become œdematously swollen, and the eyes sunken. The appetite becomes suppressed after from 4 to 6 days, although the thirst is great. Sometimes, even during the first 24 hours, the patient becomes very restless, has slight colic, and the movements of the flanks are decreased or entirely stopped. At first, the peristaltic action of the intestines is vigorous, but becomes arrested later on. In the early stages of the attack, defecation is normal, except that it is more frequent than usual. The fæces are often mixed with blood coagula the size of a pea, bean, or even that of a child's fist. In some rare cases there is at first severe, bloodless diarrhœa, combined with violent straining, in consequence of which prolapsus recti appears 2 or 3 days later, and numerous small blood coagula are sometimes found in the now thin, sanious, or entirely bloody fæces. It may happen that in exceptional and severe cases, violent straining, and severe dysenteric diarrhœa are present from the commencement. The intes-

tinal evacuations always contain coccidia. In a few instances we may find croupy membranes and a highly purulent condition of the fæces.

The course of the disease is generally acute; seldom peracute. In large herds this disease often rages for several months. In mild cases, with a normal condition of the fæces, recovery may take place in a week; or in 2 or 3 weeks if only simple bloody diarrhœa is present. In severe cases with violent initial intestinal hæmorrhage, the animal may die within 24 hours. Relapses frequently occur; but are not dangerous.

By *post-mortem* examination the small intestine is seen to have a dark brown-red colour, with dark-blood contents. The mucous membrane of the rectum shows thin bloody deposits, swelling, and petechiæ. The fæces contain numerous coccidia.

The treatment consists in feeding with dry food, as a prophylactic measure; in keeping the sheds dry and clean; and in giving sulphur, creolin, and lysol combined with mucilage or milk. As it is supposed that the infection is principally transmitted by drinking water; it is advisable to guard the wells with masonry, and to preserve the rain water as pure as possible. Similar cases, occurring among cattle, sheep, and lambs, have been observed by McFadyean in England and Moussu in France.

Pasteurellosis of Cattle.—Lignières has given this name to a plague of dysenteric character which occurred in the Argentine, and is produced by a small bacterium of the genus Pasteurella. Moussu is of opinion that this disease appears in France also (chronic diarrhœa of adult cattle).

ERYSIPELATOUS DISEASES OF PIGS.

Swine Erysipelas, Contagious Pneumonia of the Pig, and Swine Fever.

General Remarks on the Erysipelatous Diseases of Pigs.—Little was known concerning the respective differences between the epizoötic diseases of swine, until the investigations of Eggeling, Löffler, Schütz, Lydtin, Schottelius, Bang, and others, lately threw light on their respective etiology, symptoms, and anatomical changes. Even at the commencement of 1880, almost all the epizoötics of swine were included in the collective name of "swine erysipelas." The researches of the last two decades, however, enable us to divide them into the following independent diseases:—

1. *Swine erysipelas*, which is a specific septicæmia produced by a minute bacillus that manifests itself anatomically by hæmorrhagic gastro-intestinal inflammation, inflammation

of the kidneys, swelling of the spleen, and parenchymatous inflammation of the liver, heart, and muscular system.

2. *Contagious Pneumonia of the Pig and Swine Fever.*—These two disorders are frequently found combined.

(*a*) *Contagious Swine Pneumonia* is produced by the bacillus suisepticus; and, in acute cases, appears as a multiple, necrotic pneumonia; in chronic ones, as a catarrhal pneumonia confined to the anterior lobes.

(*b*) *Swine Fever* is produced by the bacillus suipestifer; and consists essentially of a croupous or diphtheritic inflammation of limited sections of the intestinal mucous membrane.

History.—The name of swine erysipelas (splenic erysipelas, swine distemper, swine typhus, wild-fire, the rose, St. Anthony's fire, petechial fever, spotted fever, spotted fire) was formerly given to several porcine diseases which externally resembled each other, only by the fact that they respectively produce redness of the skin. But as in swine, a large number of diseases, both internal and external, display the symptoms of redness of the skin, the signification of the term swine erysipelas, as usually employed, is even more vague than that of colic or hæmaturia. A thorough examination of the old literature shows, however, that the term usually denoted the infectious disease which has of recent years been more fully investigated and described as a coccus-erysipelas. In the second rank may be placed the diseases referred to as swine distemper and swine fever. After these should be ranked those following, which were formerly often included in the same category: nettle-rash (urticaria), splenic fever, purely cutaneous inflammations of erysipelatous appearance, septicæmic disorders, poisonings, possibly also cases of worm-disease (strongylus paradoxus), heat-stroke, cases of suffocation; various internal acute diseases which are accompanied by redness of the skin, as for instance, inflammation of the lungs; and probably several infective diseases, the pathology of which is not yet fully known. Consequently, there were great differences of opinion on the nature of swine measles. Apart from the occasional appearance of the view of its being a result of solanin poisoning, and that of its cause being due to an infection with the poison of typhus of man, we find that from the end of the previous century down to the beginning of the sixties of the last, the generally prevailing view was that swine erysipelas was identical with splenic fever. From that epoch arose the appellations of "anthrax erysipelas" and

"anthrax fever." Before 1860, Nicklas and others had drawn attention to the fact that swine erysipelas (measles) was not anthrax; because, as general experience proved, the eating of the flesh of measly pigs produced no ill consequences to man. This anthrax theory became absolutely untenable in the year 1865 by the researches of Brauell, who discovered the bacillus anthracis. He showed that pigs were immune to inoculations of anthrax blood; and that inoculations with the blood of measly pigs did not produce anthrax. Besides, the bacilli of anthrax were never found in the blood of pigs suffering from swine erysipelas. Also, Harms, in 1869, described swine erysipelas as an independent infective disease which consisted essentially in a severe affection of the gastro-intestinal mucous membrane, with parenchymatous changes in the liver, kidneys, muscles, and lymph glands, and which did not attack the lungs.

In 1882, Eggeling demonstrated for the first time in this connection that the general term of swine erysipelas included several independent diseases. He distinguished the following varieties: (1) *Sporadic erysipelas of the head*, which is an infective disease due to wounds, with frequent gangrenous desquamation of inflamed portions of the skin, and which resembles human erysipelas of the head. (2) *Sporadic and benign urticaria*. (3) *Epizoötic erysipelas*, which may be compared to scarlet fever of man. It is an acute exanthema which may be transmitted by inoculation, and which is distinguished by constitutional disturbance; copper-red, and spotted discoloration of the skin of the intermaxillary space, lower portion of the chest, space between the fore-legs, abdomen, and inner surface of the thighs; inflammation of the mucous membranes of the respiratory organs; œdema of the lungs; and changes in the liver and kidneys. (4) *Contagious pneumonia of the pig*, which is a form of septicæmia, and which is the most frequent and most dangerous porcine epizoötic. Its symptoms are severe constitutional disturbance, weakness, high fever, and a uniform redness of the skin, which rapidly spreads from the posterior parts of the abdomen over the whole body. The most important anatomical change observed in the disease is a hæmorrhagic gastro-intestinal inflammation, which is accompanied by great swelling of the mesenteric glands.

Löffler, in 1882, divided swine erysipelas into true swine erysipelas and contagious pneumonia of the pig. Here, Löffler's classification is the reverse of Eggeling's; for his erysipelas is

identical with Eggeling's contagious pneumonia; and his contagious pneumonia is probably included in Eggeling's erysipelas. The names introduced by Löffler have, however, been retained. His bacteriological researches have further furnished the following results. His erysipelas is caused by a very minute bacillus which closely resembles Koch's bacillus of the septicæmia of mice. It invariably kills mice by inoculation, and either kills rabbits, or makes them immune against the disease after they recover from the erysipelatous skin inflammation which is frequently produced by it. But contagious pneumonia of the pig is produced by small ovoid bacteria which greatly resemble Gaffky's bacteria of the septicæmia of rabbits. Inoculations of their pure cultures kill mice, guinea-pigs, birds, and pigs. Schütz found and described, in 1885, Löffler's erysipelas bacilli at first in the spleen of a pig which was suffering from measles and which had been brought from Baden; and later on, in pigs near Berlin. In the same year, Schottelius made similar observations in Baden. The nature of swine erysipelas has been fairly-well elucidated by the protective inoculations and researches of Lydtin, Schottelius, and Schütz. Pasteur, Cornevin, and Jensen have also studied this subject.

About the time when the differentiation between erysipelas and contagious pneumonia was made in Germany, a plague broke out in North America (1877–8) and England (1878), and later on in Sweden and Denmark; and then obtained a wide distribution also in France, Germany, Austria-Hungary, Servia, and other European countries. These enzoötics were known as "American," "English" and "Danish" swine-plague, swine-diphtheria, hog-cholera, etc. Of the relations of this foreign (American) swine-plague to the native (German) contagious swine-pneumonia nothing was till quite recently known with certainty, as the views of the bacteriologists were very often contradictory. It was for the first time by the investigations of Bang, Schütz, Smith, Salmon, Jensen, and lately (1898), more especially of Preisz, that the difficult question has been cleared up. Accordingly, contagious swine-pneumonia and swine-fever are two entirely distinct infective diseases, produced by separate bacteria (bacillus suisepticus and suipestifer). These diseases appear sometimes alone, and sometimes combined with one another. It more especially happens that very often contagious swine-pneumonia appears as an infection secondary to that of swine-fever (Preisz). This last fact throws light on the contradictions in the bacterio-

logical views taken of the diseases; as well as on the great complexity and frequent agreement of the anatomical and clinical conditions. In America swine-fever appears to have formerly prevailed; in Europe, and especially in Germany, contagious swine-pneumonia seems to have held the more prominent position. At present, both in America and in Europe, both diseases prevail, side by side, or in actual combination.

I.—SWINE ERYSIPELAS (*Rhusiopathia suis—Rouget du Porc*).

Bacteriology.—According to the researches of Löffler, Schütz, and Schottelius, the bacteria of swine erysipelas (bacillus rhusiopathiæ suis), are very slender rods from 1 to 1.5 μ in length, and can be seen with exactness only with the aid of a bacteria microscope (oil immersion lens and Abbé's illumination apparatus). These bacilli are found in the blood, yet comparatively sparsely placed between the red blood corpuscles, and within the leucocytes. They are most numerous in the spleen, kidneys, and liver. This is shown in stained sections of these organs. They may easily be cultivated on nutritive gelatine at ordinary laboratory temperatures. In flat cultures they form in from 2 to 3 days, under the surface of the gelatine, clustered, hazy, bluish-grey patches of turbidity. Stab cultures in gelatine have the aspect after from six to ten days, of a fir tree or of a brush for cleaning glasses;* the surface of the gelatine remaining quite smooth. The erysipelas bacillus thrives better in neutral or slightly alkaline broth; but not on potatoes.

The researches of Jäger, Petri and others, have given the following results regarding the effect of external influences on the erysipelas bacilli. The bacilli are destroyed by slow drying, in from 50 to 80 hours; by water at 115° Fahr. in 20 minutes; by water at from 131° Fahr. to 158° Fahr. in 5 minutes; by water at 194° Fahr. in 2 minutes; by dry air at 115° Fahr. in 3 hours and a half; and by air at from 37½° Fahr. to 46½° Fahr. in 13 days. They are speedily killed by quicklime (1 to 2), chloride of lime (1 to 100), a hot solution of soda or of potash (1 to 100), carbonate of soda (1 to 100), sulphate of iron (1 to 3), permanganate of potash (1 to 20), sulphate of copper (1 to 400), and by all the stronger antiseptics, such as corrosive sublimate and creolin, etc. A concentrated solution of common salt, like

* In this brush, the bristles radiate round a stem, with which they form a angle of 90°.—Tr.

that used for pickling meat, takes 26 days to kill the bacilli; and brine, 7 days. Ordinary boiling and roasting will not kill all the bacilli in pieces of meat exceeding 2½ lbs. in weight; but two hours and a half boiling (not roasting) will destroy them in smaller pieces of meat. The erysipelas bacilli in infected meat are destroyed only with great difficulty by pickling and smoking. They remain unchanged in ordinary pickled meat for several months, indeed, even for 6 months; and in smoked ham, for 3 months. We can see from these experiments that the ordinary methods of preparing pork are not sufficient to kill the bacilli in meat. Saturated solutions of boric acid, tannic acid, arsenic, benzine, chloride of zinc, and carbolic acid in spirit, do not accomplish that end, even in 48 hours. The bacilli lose their virulence in sea water after 12 days, but remain alive in it for over 24 days.

Inoculation (cutaneous and subcutaneous) succeeds most readily in white mice, which die after an illness of from 2 to 4 days under symptoms of septicæmia of mice. Rabbits die in from 5 to 9 days, or become affected with a local erysipelatous dermatitis, after recovering from which they are found to be immune to further infection. Pigs, which in other cases are found hard to infect artificially, also die from the results of inoculation, with the characteristic symptoms of erysipelas, and manifest bacilli in all their organs; so do pigeons (death occurs after from 3 to 4 days). According to Cornevin and Kitt, the inoculation is negative in guinea-pigs, fowls, geese, ducks, ordinary rats, dogs, cats, mules, asses, horses, cattle and sheep, all of which must be considered to be immune to bacillar erysipelas. On the other hand, isolated cases of infection have been noticed in human beings (see Vol. II., p. 243).

Bacillus Murisepticus.—The bacillus murisepticus, which was formerly regarded as a distinct variety (septicæmia of mice), is, according to Prettner, identical with the erysipelas bacillus; and, therefore, to be discarded as a specific bacillus. It represents a weakened variety of the erysipelas bacillus; as the latter, in the body of the mouse, is found to lose its virulence for pigs.

Pathogenesis.—According to Pasteur, Schütz, Schottelius, Lydtin and Cornevin, the bacilli enter the body chiefly by the digestive canal. Jensen considers that they can also get into the system by means of even very minute wounds of the skin. According to Fritsche, experimental animals have been infected by the erysipelas bacilli, even through the unshaven

skin (introduction through the hair-follicles !) Buchner's supposition that the bacilli can be absorbed from the inspired air by the lungs appears impossible; for Cornevin has proved that they lose their virulence by being dried. They rapidly increase in number after gaining entrance into the blood, and especially directly after death. Their pathogenical effect depends on the excretion of a toxin, which chiefly acts on the nervous system, the muscular system, and on the cells of the parenchyma of the large glands. Only by this fact are we able to account for the paralysis, grave cerebral disturbance, and anatomical changes in the large glands.

As a rule, one attack confers immunity.

Occurrence and Dissemination.—Bacillar erysipelas is an infective disease which is widely spread and which occurs enzoötically and even epizoötically, probably in most of the countries of Europe. Its dissemination corresponds to that of the bacilli of erysipelas, which appear to thrive particularly well in stagnant water. For instance Löffler states that it is constantly found in the Panke [a small stream that runs into the Spree at Berlin]. Like anthrax, erysipelas is often stationary. It was formerly restricted in Bavaria to the districts of the Danube, and was entirely unknown in Southern Bavaria (Kitt). Lydtin states that the disease tends to become enzoötic chiefly in valleys and low-lying plains which have slow-flowing streams, and heavy, damp clay soil; and that sandy and granite soils are comparatively free from it. It occurs chiefly during July, August and September, and sporadically during winter. Sultry summer heat and thunderstorms seem to promote the development of the infective matter. According to Lydtin, the majority of cases in Baden have been observed on small farms which had insanitary pig-sties; in places where great numbers of pigs were kept; and where distillers' wash was used as food. It is usually supposed that sties which are damp, ill-ventilated, and polluted with the fæces of pigs, are especially favourable to the development of the disease. The same remark applies to bad, damaged food, which formerly was wrongly regarded as the direct cause of swine erysipelas.

Pigs are least predisposed to the disease during the first months of life. Cases occur most frequently among animals of from 3 to 12 months old; and rarely after the age of 3 years. Sucking-pigs do not contract the disease by drinking the milk of infected sows; though Koubasoff states that the

bacilli pass into the milk. It has long been known that the respective susceptibility of different breeds of pigs varies greatly. Thus, Hable found that in Austria, imported breeds become more readily affected than indigenous swine. Lydtin states that in Baden, English and half-bred English (Suffolk) pigs become earliest affected; then Polish-China pigs, and that Yorkshire pigs and their cross-breeds were less susceptible. The common country pig was least susceptible of all; for which reason it is not suitable for inoculation experiments.

Swine erysipelas is chiefly a miasmatic disease, and thus differs from swine fever and contagious swine-pneumonia, which, as a rule, can be spread only by direct infection. The bacilli of swine erysipelas, similar to those of anthrax, become further developed outside the animal body (ectogenous development). Also, as with anthrax, an indirect infection takes place principally by means of the water and soil of the permanently infected districts. Direct infection may occur, but only by the absorption of infected excrements or of infected flesh. Transmission by the air does not take place. Hence, healthy pigs which live close to infected sties, do not take the disease, if they are prevented by partitions from coming into direct contact with the sick animals, even when the air is allowed to pass freely from one sty to the other. Consequently, we must look upon the contagium as fixed.

The disease is often spread by the sale of the flesh of erysipelatous pigs, which were necessarily slaughtered; whereby the disease has sometimes been spread over a whole neighbourhood; or, by means of hawking of the flesh from house to house, it may be simultaneously distributed over several villages. This has been noticed by many observers, and also confirmed experimentally. Swine erysipelas is, however, chiefly distributed by consumption of the refuse of slaughter-houses and kitchens; by the water in which the flesh has been washed; by the illegal sale of the flesh of erysipelatous pigs; by the use of unclean drinking vessels; by contamination of the drinking water; by rinsing of the slaughter utensils; and by the butcher himself. Lydtin states that in a certain village, swine erysipelas was spread along the banks of a brook, into which the waste water of the neighbouring inhabitants went. Cornevin points out that the disease may be spread by giving pigs the brine in which infected pork has been salted.

Presence of Erysipelas Bacilli in the Intestines of healthy Pigs.—According to Olt, the bacillus of erysipelas occurs normally in

the intestine of the pig. He proved, by the examination of pigs from different localities of Germany, that in very healthy pigs, as in the almost constantly occurring entozoic ulcers of the cæcum and colon, and the follicular entozoic plugs, the erysipelas bacilli are found to vegetate. Bauermeister has also demonstrated the normal occurrence of the erysipelas bacilli in the tonsillar plugs and tonsillar secretion of healthy pigs. Accordingly, in an enzoötic spread of erysipelas, its causative agent need not be taken from the ground, as the erysipelas bacilli are normally present in the intestines and tonsils of healthy pigs. It is plain that the altered conditions of life in pigs turned out to pasture, and injurious influences of every kind, predispose to the erysipelas disorder; and that the infection is introduced solely by the bacilli present in the intestine. On this account, Olt, having regard to the distribution and mode of existence of the bacillus of erysipelas, considers the campaign against it by means of veterinary inspection regulations, disinfection of styes, etc., as hopeless. Jensen has confirmed the presence of erysipelas bacilli in the tonsils and intestines of healthy pigs.

Symptoms.—After a period of incubation of at least 3 days, the disease usually begins suddenly and violently. The animal refuses its food; sometimes vomits or makes efforts to vomit; has a very high temperature (up to 109·4° F.); manifests severe nervous disturbance; is very weak, torpid, sleepy, and indifferent to its surroundings; and tries to hide itself under its bedding. The hind-quarters become weak and paralysed. Muscular spasms and grinding of the teeth are sometimes observed. At first there is constipation; the mucous membrane of the eyelids is of a dark-red or brown-red colour; and the eyelids are sometimes swollen. Usually, a couple of days after the first manifestations of the symptoms, or even from the very commencement of the attack, spots appear on the thin parts of skin, such as the region of the navel, lower surface of the chest, perineum, inner surface of the thighs, ears and throat. These spots, which at first are bright red and about the size of a man's hand, become, later on, dark red or purple, and soon unite into large irregularly-shaped patches. As a rule, they are neither painful to the touch nor prominent, but sometimes show a slight inflammatory swelling. The skin of the red spots, especially of the ears, may suffer from an eruption of vesicles, and may even slough. This spotted redness of the skin may be very slight in severe cases, or may appear only immediately before, or even after death. At other times it may be entirely absent, or may become spread over the whole body. Diarrhœa also sets in, and the fæces become thin, mucilaginous, and sanious in a few instances. Towards the end, respiration becomes greatly accelerated, general cyanosis (œdema of the lungs) supervenes,

and death takes place, usually on the third or fourth day of the attack, with increased general weakness and considerable fall of temperature (down to 98·6° F. or less). When the disease is very severe, the animal may die in 24 hours. Sometimes the disease takes a week or longer to run its course.

Different clinical forms of Epizoötic Erysipelas.—Jensen considers that this disease, instead of being uniform in its clinical aspect, manifests itself in different, well-characterised forms, between which transition stages sometimes appear. Jensen distinguishes the following types: 1. Erysipelas in the stricter sense; 2. Nettle-fever (urticaria); 3. Erysipelas without redness of the skin (rouget blanc); 4. Diffuse erysipelatous necrosis of the skin; 5. Erysipelatous endocarditis (endocarditis verrucosa bacillosa). In addition to these other chronic forms appear.

1. **True erysipelas** is the usual form of erysipelas, and is the one we have just described.

2. **Swine urticaria** is said to be a mild form of erysipelas, the character of which is due partly to the difference in the manner of infection (infection of wounds of the skin), partly to the slight degree of virulence of the contagium, and to the temporary diminished susceptibility on the part of the animal. It is said that the bacilli of erysipelas have been found in cases of urticaria (Jensen and Lorenz). Consequently, these two authors maintain that swine urticaria, which was formerly described among cutaneous affections as an independent, benign, infective disease, must be included in erysipelas. We, however, agree with Schindelka that no valid reason exists for referring all cases of urticaria of pigs to erysipelas; because an eruption of urticaria, as in other domestic animals, may certainly occur independently of erysipelas. The observations of Jensen and Lorenz, provided that they will be confirmed by further researches, are very important; for they appear to show that, apart from ordinary urticaria, there is a symptomatic nettle-rash which may be regarded as a form of epizoötic erysipelas. According to Jensen, the symptoms of this form are as follows: The disease, as a rule, begins rather suddenly, by the animal ceasing to feed, showing high internal temperature from 106° to 107° F. and even to 108·5° F., and being extremely thirsty. The fever increases during the following two days, and the patient becomes languid, stiff, and so weak that it appears as if paralysed. One or two days after the manifestation of the first symptoms of the disease, more or less round, raised spots appear all over the body, and especially on the loins, outer surface of the thighs, neck, and shoulders, which are parts of the body that in ordinary erysipelas usually suffer from no cutaneous changes. These spots increase rapidly in size and number, and become dark-red, violet, or more or less black. On the third or fourth day, the wheals are sharply defined, and are generally square or rhomboidal, but may be round or oblong. They are 1 - 2½ inches in diameter, or even larger; are raised above the healthy skin only a few millimetres as a rule; and often run together so as to form large irregular, angular spots. They are of a dark-red colour, and number from 50 to several hundreds. In very slight cases, we may find on the entire skin only a single, square-shaped red wheal. After the

development of the wheals, the fever increases and the animal begins again to feed. In a few days, the wheals disappear with desquamation of the epidermis ; or in severe cases, with sloughing of the skin and the formation of a dark, leathery crust. The whole duration of the disease varies from 8 to 12 days. The mortality is very small ; as the course of the disease is almost always mild. Chronic endocarditis is a rare sequela. Urticaria and the ordinary form of erysipelas often appear simultaneously, or one after the other, in the same drove of pigs. Inoculations with pure cultivations of the bacilli of urticaria and with portions of the tissues of infected animals have almost always produced a negative result in pigs (Bang and Jensen.)

Schütz has found, in conformity with his experiments on erysipelas inoculation, that after the injection of one and the same culture of erysipelas bacilli, there appeared in one pig a brick-coloured patchy eruption, and in another true erysipelas. Whether the former or the latter occurred, depended on the breed or individuality of the pig. Whilst after injection of a pure culture of erysipelas bacilli, an eruption of brick-coloured patches appeared in the case of pigs of the ordinary breed ; the English, and especially the English of the highly-bred types, sickened with the symptoms of erysipelas. Schütz has also very often observed that the sickness of the artificially infected pigs began in the form of a brick-patch eruption, and passed after a short time into that of erysipelas. The bacilli of the brick-patch eruption do not preserve their small toxic powers. Schütz has made them so toxic by a number of transmissions through highly-bred pigs, that at last every pig, even the ordinary country breeds, succumbed as soon as the bacilli were injected under the skin.

By reason of these facts, the same rules for veterinary inspection should be applied to the cases of the brick-patch eruption as to those of erysipelas, with this limitation : that the flesh, after the removal of the altered portions, may be placed in the open market, as the bacilli are found only in the affected portions of the skin.

3. **Erysipelas without redness of the skin** (French, *rouget blanc*) is a rare and very acute form of erysipelas, from which the sufferer dies too quickly for the redness of the skin to become developed.

4. **Diffuse necrotic erysipelas of the skin** begins, according to Jensen, with fever and with the appearance of irregular, flat, red swellings, about the size of a man's hand, on the neck, back, sides, and joints of the shoulders. These swellings tend to become confluent, so that almost all the skin on the upper part and sides of the animal is swollen and reddened. The red gradually becomes deeper in colour, or may turn into deep violet or nearly black ; and the attacked parts of the skin lose their sensitiveness and warmth, become hard, dry, and necrotic, and slough with the formation of a line of demarcation. The ears and tail often rot off. The resulting cicatricial tissue is firm, more or less like cartilage, and subsequently contracts so much that the affected part becomes deformed. Some of the sufferers die at a very early stage, and others linger on for 1 or 2 months. Many pigs are slaughtered on account of their having become permanently disfigured. The cutaneous necrosis varies in size from spots that are as large as a man's hand, to patches which may extend nearly over the entire body. Bacteriological examination

shows that numerous erysipelas bacilli are present in the lymph spaces of the cutis directly under the epidermis.

5. **Endocarditis of erysipelas** has been carefully studied by Bang. It often begins acutely with the ordinary symptoms of erysipelas, and is followed by a latent period, during which the animal apparently recovers ; but subsequently becomes affected with a well-marked, visible, cardiac affection, which generally lasts for a week or two. In a few cases, the pigs die from apoplexy. The symptoms are : loss of appetite, depression, persistent lying down, shortness of breath, slight cough, and redness of the skin, in varying intensity and extent ; although not to such a high degree as in acute erysipelas. After this, we find palpitation of the heart, greatly accelerated action of that organ, and sometimes endocardial murmurs. The temperature is often increased and the animal dies with symptoms of cardiac paralysis. In a few cases, we may observe paralysis of the hind-quarters. On *post-mortem* examination, the left side of the heart is usually found to be affected ; the right, less frequently. The ostium is almost always plugged up, and the mitral valve is the most frequent seat of attack. The valves are swollen and thickened, and show verrucose vegetations with deposits of thrombi, in which large numbers of erysipelas bacilli can be found. According to Bang, endocarditis of erysipelas is not rare. For instance, out of 10 pigs which had passed through an acute attack of erysipelas, 9 died of endocarditis within 3 months. The fact of slaughtering for food animals which have apparently, but not really, recovered from epizoötic erysipelas, greatly furthers the spread of the disease. Bang therefore recommends that the segregation of pigs recovering from erysipelas should be continued after they have apparently become quite healthy ; or that they should be slaughtered and the owners indemnified.

6. **Other chronic forms of epizoötic erysipelas.**—Cornevin, Hess, and others have described chronic cases which have become developed from an acute attack. The aspect of chronic erysipelas differs essentially from that of the acute form. Although the appetite remains good after the acute attack has subsided, the animal becomes emaciated, the belly is drawn up, and there is weakness, diarrhœa, and shortness of breath. The patient suffers from a chronic state of ill-health, which may last several months, and which, it is said, may be followed by symptoms of so-called " scurvy " or " bristle-rot," such as hæmorrhages in the bulbs of the hair, tendency of the bristles to fall out, and bleeding and ulceration of the gums. It is said that, besides these symptoms, we may sometimes find in certain organs, especially with inoculated animals, local changes such as enteritis, endocarditis verrucosa, and arthritis fungosa, with resulting chronic lameness and deformity of the skeleton. It is also stated that redness of the skin can be observed at the beginning and during the exacerbations of almost all these chronic cases, and that it is an infallible sign of approaching death.

Prognosis.—The ordinary kind of epizoötic erysipelas has a mortality of from 50 to 85 per cent. In Germany, in the years 1897 and 1898, the loss reached from 93 to 95 per cent. ; in 1899

and 1900, 89 to 91 per cent.; and in 1902, 83 per cent. (of 52,000 affected pigs). Lydtin states that the losses from it in Baden were between 50 and 75 per cent. It is stated that during 1873 in Denmark and Schleswig 87 per cent. of all the pigs affected with erysipelas died. In Hungary, in 1899, of 19,000 affected pigs 10,000 died (53 per cent.); in 1900, of 21,000, 15,000 died (72 per cent.). In any case the prognosis is always very unfavourable, as we may judge by the fact that the financial losses caused by epizoötic erysipelas and contagious swine-pneumonia in different countries far exceed those from every other animal disease. Thus the losses in the Grand Duchy of Baden, within twenty years (1874–1893), amounted to about £150,000 (135,000 cases of swine erysipelas); of which in the year 1884 alone, for example, it reached about £13,500 (12,000 erysipelas pigs—3 per cent. of the entire value). Some localities in Prussia are specially marked by the endemic prevalence of swine erysipelas. Mehrdorf calculated the annual loss in the Königsberg circuit alone at from £100,000 to £159,000. Steffen reckoned the annual loss in the municipal area of Magdeburg at from 15,000 to 20,000 pigs. In that of Oppeln the disease, according to Schilling, caused losses equal to a fifth of the whole of the taxes. A tenth in the plague-stricken villages, and one-half in the infected sties, of all the pigs contracted erysipelas; the annual losses in this locality amounting to £20,000. In the Kolmar circuit, according to Heyne, 2,600 pigs out of a total of 17,000 died of erysipelas within a year. During 1891 in Württemberg, 21,500 pigs out of a total of 350,000 suffered from erysipelas (6 per cent.), with a loss of £22,000. Zschokke and Hess state that the yearly loss from it in Switzerland amounts to many thousands of pounds sterling. The value of the pigs which died of erysipelas in Brittany during 1881 was £120,000. In France during 1887, out of 19,000 infected pigs, more than 15,000 died; thus showing a loss of £20,000. The German Imperial Board of Health puts the yearly loss of the German Empire from swine erysipelas at £225,000.

The hopeful cases are those which live over 4 days. According to Jensen, the prognosis of the urticarial form is very favourable. [With regard to the beneficial results of the serum treatment, see chapter on Inoculation.]

Anatomical Conditions.—In *post-mortem* examinations of cases of the ordinary form of epizoötic erysipelas, we find signs of septicæmia without any well-marked morbid condition of

separate organs. This septicæmia is a general infection which produces hæmorrhagic and diphtheritic gastro-enteritis, considerable swelling of the lymphatic system, hæmorrhagic or parenchymatous nephritis, acute swelling of the spleen, parenchymatous hepatitis, and myositis.

1. The *hæmorrhagic gastro-enteritis* consists at first of excessive inflammation of the mucous membrane of the stomach in the region of the fundus. The mucous membrane shows a dark-red discoloration, which is partly diffuse, partly in spots; suffers from cloudy swelling; often has eminences on its surface; is covered with a viscid layer of mucus; and may even have superficial scabs. The glands of the mucous membrane are inflamed (gastritis glandularis). The intestinal mucous membrane is swollen, especially on the top of the folds, narrow parts of the small intestine, and in the neighbourhood of Peyer's patches; is covered with reddish mucus; infiltrated with hæmorrhages; and sometimes shows superficial scabs. Less frequently, circumscribed parts of the mucous membrane of the cæcum and of the anterior part of the colon suffer from a diphtheritic affection.

2. *The solitary follicles and Peyer's patches* present, throughout, medullary swellings in the form of prominent raised patches of the size of a grain of millet to that of a lentil. Sometimes they are infiltrated with hæmorrhages and surrounded by a red ring. We very frequently notice ulceration and cicatrisation of the solitary and agminated follicles. The mesenteric glands become more swollen than the other glands of the body; are of a dark-red colour; and show softening. The surface of their section is dun-coloured with interspersed dark-red spots; and the paraglandular tissue is hyperæmic, and infiltrated with hæmorrhages.

3. *The hæmorrhagic nephritis* is distinguished by enlargement of the kidneys, and by the kidneys assuming a grey-red colour. The medullary layer is generally of a very dark red, and the cortical layer is infiltrated with blood points and widened out. In slight cases, we find only a parenchymatous inflammation (cloudy swelling) of the kidneys. Frequently, catarrhal nephritis occurs as a complication.

4. *Acute swelling of the spleen* arises in consequence of an acute, severe hyperæmia with great increase of the cellular constitutents of the spleen (new formation of splenic cells), in which case the spleen is enlarged, but not softened as in anthrax. It is, on the contrary, tense to the touch, as the

capsule is considerably stretched. The pulp on the surface of a section is purple, moderately soft, and free from hæmorrhages.

5. *The parenchymatous hepatitis* consists of a cloudy swelling and enlargement of the liver, in which the surface of sections has a greyish-brown colour, and the acini are widened out. The muscles are grey in colour, soft, flaccid, watery, shiny, are sometimes infiltrated with hæmorrhages, and look as if they had been boiled. The myocardium shows similar spotted changes, and subendocardial hæmorrhages.

In the abdominal cavity, thoracic cavity, and pericardium, we sometimes find small quantities of an orange-coloured clear fluid, which may be mixed with flaky coagula ; and punctiform hæmorrhages under the serous membranes, especially on the auricles. We rarely meet with severe hæmorrhages in the brain, or in the dorsal and lumbar portions of the spinal cord. The congested parts of the skin are somewhat discoloured after death. The lungs remain unchanged, or at most exhibit a *post-mortem* œdema. By microscopical examination, the bacilli are found everywhere in the body, especially in the spleen and kidneys, and to a less extent in the blood.

[English veterinary surgeons, as a rule, regard the occurrence of more or less luxuriant vegetations on the valves of the heart (chiefly the mitral), to be so frequent in this disease, as to be almost diagnostic. It would appear that this endocarditis is not nearly so common in Continental cases.—TR.]

Diagnosis.—Apart from the demonstration of the bacilli, the diagnosis of bacillar erysipelas depends essentially on the presence of swelling of the spleen, gastro-enteritis, nephritis, affection of the lymphatics, and absence of inflammatory changes in the lungs. In doubtful cases, we recommend the inoculation of white mice or pigeons with the blood or with small pieces of the spleen under the skin of the back or on the chest. When erysipelas is present, inoculated mice become affected in 24 hours. They are depressed and arch their backs ; the coat stands on end ; the breathing is accelerated ; and the mucous membrane of the eyes discharges a mucilaginous, glassy secretion. Death takes place on the fourth day after inoculation. Inoculated pigeons die in 3 or 4 days. The erysipelas bacilli can be easily found in the blood of the inoculated animals. For more exact diagnosis, Johne recommends the preparation of a stab culture in the following way. The spleen of a dead pig is first of all washed in a watery solution

of corrosive sublimate, 1 to 1,000, and torn asunder in the middle; a platinum needle which has been sterilised by being made red-hot and allowed to cool, is introduced into the middle of the pulp of the spleen 1½ or 2 inches deep, and is then stuck about the same depth into the meat peptone-gelatine. The test-tube containing the nutritive gelatine is held with its mouth downwards, and the cotton-wool plug is only removed directly before the inoculation. After 5 or 6 punctures have been made with the platinum needle and after the cotton-wool plug has been replaced, the test-tubes are allowed to stand for about 4 or 5 days in the ordinary temperature of the room, after which time, the characteristic slender cultivations, which radiate outwards like a pine tree, or like a chimney-glass brush, form in the nutritive gelatine.

Differential Diagnosis.—The following are the principal diseases which have to be distinguished from epizoötic erysipelas :—

1. *Contagious pneumonia of the pig and swine fever* differ essentially from epizoötic erysipelas by their bacteriological results (ovoid bacteria), and by the peculiar changes in the lungs and intestines. According to Cadiot, the two diseases may also be differentiated by inoculation. If, after inoculating a pigeon and a guinea-pig, we find that the former dies, and the latter remains unharmed, we may conclude that the disease is erysipelas ; and contagious pneumonia, if the reverse of this takes place.

2. *True cutaneous erysipelas*, which is an infective disease, and arises from wounds chiefly on the head, is characterised by an inflammatory, painful skin affection, with tendency to gangrene (sloughing of circumscribed pieces of skin, and of the tips of the ears), and is localised principally on the skin of the head.

3. *Heat apoplexy*, which occurs especially in very fat pigs during transport when the weather is hot, and which causes a purple discoloration of the skin on account of vascular engorgement. The *post-mortem* appearances are essentially those of suffocation.

4. As a rule, *anthrax* occurs very rarely in pigs, and then it is nearly always localised as gloss anthrax, or as anthrax angina, in which case, there is difficulty in swallowing, dyspnœa, and carbuncular swelling of the skin and of the intermaxillary space. The presence of the bacilli anthracis, which are 10

times larger than the bacilli of erysipelas, confirms the diagnosis of anthrax.

5. The nettle rash (urticaria) is, in contrast to the "brick eruption," not infective. It occurs sporadically and runs a very benign course.

6. Erysipelas may be confused with *traumatic erythemata*, as for instance, blows inflicted on driven pigs, and injuries of the teats or udder of a sow caused by sucking-pigs.

Therapeutics.—The course of epizoötic erysipelas is so rapid that medicines are generally of no avail. In former times it was the custom at the beginning of the attack to give an emetic, which, we are unanimously assured, had a good result. Berner reported in 1858 that he cured 75 per cent. of all his cases by this treatment, the favourable effect of which is to be ascribed to the removal of the infective matter. On similar grounds, when treatment is possible, we can strongly recommend the administration of large doses of calomel (45 to 75 grains), in order to thoroughly disinfect the stomach and intestines by the perchloride of mercury that is formed from the calomel. Creolin is also considered to be an effective remedy at the beginning of the disease. Obel recommends that 2 or 3 liqueur glasses of spirit (whisky, for instance), should be given later on in a quart of milk. With regard to the curative influence of serum-inoculation, see Protective Inoculation, p. 238.

Prophylaxis is much more important than treatment. As prophylactic measures, inoculation and veterinary inspection come specially into consideration in fighting erysipelas (Inoculation will afterwards be dealt with in a special chapter). Plague-inspection regulations have already been, to a certain extent, enforced against erysipelas in various countries. In Germany, by an order of the State Chancellor of September 8, 1898, the rules contained in paragraphs 9 and 10 of the State Regulations in reference to cattle plague, to which we have already referred, are also extended to contagious swine-pneumonia and swine-fever. More rigid regulations regarding veterinary inspection have, in accordance with the draft given below, been brought to a comparative state of uniformity in most parts of Prussia. Further, by the alteration and extension of the regulations relating to the cattle plague, the local enforcement of the inoculation of pigs, placed in a position of danger, has

been secured. The period over which the warranty extends is three days, according to the German civil law.

Veterinary Police Regulations.—The Royal Technical Commission on Veterinary Matters in Prussia published, in 1896, the following Police Regulations for erysipelas:

1. The duty of notification on the outbreak of erysipelas.
2. When the appearance of a case of the disorder has been certified by the official veterinary surgeon, the police authorities can, in the event of a new outbreak, apply the necessary protective regulations to the infected locality, without again calling in the official veterinary officer. The veterinary surgeon has, however, to give directions, at stated intervals, regarding the carrying-out of the prescribed regulations, and, more especially, to supervise the processes of disinfection in the infected localities.
3. The pigs cannot be taken to or removed from the affected farmyard, so that the animals which still appear to be healthy, or those which show only the initial symptoms of the disease, may not be conveyed from an infected farmyard for the purpose of slaughter.
4. In the case of a general spread of erysipelas an order may be made that the removal of pigs from the infected locality, or the transit or transport of pigs from a distance through the infected locality, can be carried out only in waggons under police supervision.
5. The local police authorities can close the cattle-markets in the infected localities; and also forbid the driving of pigs to or from public markets.
6. The bodies of the dead or slaughtered erysipelatous pigs, as well as their refuse, have to be thoroughly destroyed.
7. After the removal of the cases of disease, disinfection of the infected sheds, the dung-heap and other surroundings which may have been contaminated by the infective matters, must immediately be carried out.
8. When erysipelas appears in a driven herd, or among pigs in process of transport, the further despatch of the animals must be forbidden. But arrangements can be made for their conveyance by railroad or transport waggon for the purpose of immediate slaughter.
9. The plague can be declared to be at an end, and the preventive rules allowed to lapse by the police authorities, when an interval of five days has passed after the last case, and the requisite disinfection has been carried out.

Protective Inoculation against Erysipelas.—With regard to the veterinary police campaign, inoculation has, according to the experiences of the last decade, proved itself the most valuable prophylactic agent in the extinction of erysipelas. It is, however, but the necessary inoculation and the precautionary inoculation which here enter into consideration; *i.e.*, the inoculation in infected sheds, and in the neighbourhood of infected localities. On the other hand, we do not enter on a discussion of protective inoculation in the narrower sense, *i.e.*,

of inoculation in absolutely healthy sheds and localities, as well as in plague-free periods, against the danger of the spread of erysipelas.

Of inoculation methods and preparations, there are at present four in use.

1. The method of Pasteur.
2. The method of Lorenz.
3. The Susserin.
4. The Porkosan.

1. The **Pasteur method of inoculation.**—In 1882, Pasteur first introduced protective inoculation against the erysipelatous disease of swine. In 1884 he had improved his methods, and subsequently provided an inoculation-matter, which was obtained by passing the erysipelas bacilli through the bodies of rabbits and a subsequent stage of growth in cultures. The inoculated animals were, according to Pasteur, but slightly ill, while they remained immune to erysipelas for a year. The process of inoculation consisted in first inoculating the pigs at an age of from 9 to 16 weeks, and with a weaker form of inoculation-matter (primary vaccination). They were then inoculated with a stronger preparation, about 10 to 12 days afterwards (secondary vaccination), in the subcutaneous connective tissue of the inner aspect of the thigh. Protective inoculations with the Pasteur vaccine (which may be obtained from Stuttgart) have also been carried out on great numbers of pigs in France, Baden, Prussia, Hungary and Switzerland. Researches in Baden (Lydtin) have shown that the Pasteur form of protective inoculation conferred a certain degree of immunity on the inoculated animals, but that the dangers of inoculation were not inconsiderable—as the inoculated animals immediately infected the healthy ones, so that the disorder was spread more widely, while the subjects of inoculation became seriously ill, and might even succumb to its effects—4 per cent. of all those experimented on in that series of researches died. Experiments made in Switzerland with the Pasteur inoculation-matter, also gave unfavourable results (Hesz, Guillebeau). Better effects have, on the other hand, been obtained in Hungary (Hutyra). The most important of the inoculation researches are as follows :—

In *Baden*, inoculation experiments were carried out during the years 1885 to 1887, under Lydtin's supervision. In 1885, 237 pigs of different breeds were employed. They were from 9 to 19 weeks old; weighed from 24½ to 103½ lbs., and were distributed in 15 stations over the whole country;

so as to give due consideration to the respective influences of climate, soil, farming, feeding, and absence or presence of the disease in localities. The inoculation was carried out by an assistant of Pasteur in the following way. The pig having been placed on its back with its hind-legs held asunder, about 1½ mins. (0.1 c. c.) of fluid contained in a Pravaz syringe, which had been supplied by Pasteur, was injected subcutaneously on the inner surface of the thigh. Of the 237 animals, 119 were inoculated and the remaining 118 were kept as control animals. The inoculation of the first lot was made at two different periods, at first with a weak material (*premier vaccin*), and 12 days afterwards with a stronger material (*deuxième vaccin*). Twelve days subsequently, 60 out of the 119 "protected" animals, and 60 out of the 118 "control" animals, were subjected to the influence of the infection; some of them receiving injections of Pasteur's *virus fort*, and others being fed on the flesh of pigs which had died from erysipelas. The respective results of the protective inoculation, and of the transmission of the disease by the *virus fort* or by infected flesh, were as follows: (1) Out of the 119 "protected" animals, 80 per cent. reacted to the first inoculation by a rise of temperature; 15 per cent. showed all the symptoms of erysipelas; and 5.4 per cent. died from erysipelas. By the second inoculation of the remaining 113 pigs, 46 per cent. showed a considerable increase of temperature; 7 per cent, manifested well-marked erysipelas; but there was no case of death. (2) Out of the 118 "control" pigs, 62 per cent. became infected by contagium received from the inoculated animals after the first inoculation and 1 died of erysipelas. After the second inoculation, 36 per cent. became infected with a rise of temperature; 4 showed well-marked symptoms of erysipelas; and 1 died from erysipelas. The effects of the *virus fort* were as follows: (*a*) Out of the 60 "protected" animals there was a slight rise of temperature among 19, and 4 suffered from a mild attack of the disease. (*b*) Out of the "control" animals, 61 per cent. became seriously affected and 40 per cent. of these showed well-marked erysipelas. In all, 26 per cent. of the "control" animals died of the disease. In Baden, 26 out of 462 inoculated pigs (5.6 per cent.) died in 1886; but in 1887, only 2 out of 152 inoculated pigs (1.3 per cent.) succumbed.

In *Switzerland*, Hess inoculated with Pasteur's vaccine 46 pigs, of which 11 became violently affected with erysipelas by the first inoculation; 3 of them had to be slaughtered; 6 remained sickly; and only 2 made a complete recovery. No symptom of the disease was apparent directly after the second inoculation; but, later on, 9 became gradually sickly, and signs of enteritis, endocarditis, and arthritis were found on *post-mortem* examination. Consequently, out of 46 pigs only 26 remained healthy; and 15 of the remaining 20 became chronically affected. Hess and Guillebeau assume that these untoward sequelæ were caused by Pasteur's vaccine having become contaminated. They state that their confidence in it was shaken. Hess found that the inoculation was very liable to cause the death of old and fat pigs from erysipelas; and that the inoculation of pigs more than 4 months old was hazardous.

In *Prussia*, Jakobi of Obornik first carried out protective inoculations. He reports that the loss amounted to 50 per cent. with old pigs. On the other hand, there were only 14 deaths among 1,036 pigs which were under 16 weeks old and which were inoculated according to Pasteur's method during the years 1888 to 1894. Salchav (1897) inoculated 178 pigs

without any deaths, and with favourable results, but unfavourable ones were obtained by Ehlers (50 per cent. of fatal inoculations of old pigs); with Peschke 15 per cent. caught the infection of erysipelas in spite of the inoculation; with Mehrdorf, there were 50 per cent. of fatal inoculations among 300 pigs, within 4 weeks; with von Lojewski, 7 per cent. of fatalities among 1,000 pigs; with Willutzki, 15 per cent. of fatal cases; Bermbach, Frick, etc. Röder had favourable experiences in Saxony in 1894.

In *Hungary*, Hutyra states that out of one million young pigs which were inoculated during the years 1889–1894, only 8,216 died (0.75 per cent.) in consequence of the inoculation. From this he concludes that Pasteur's protective inoculations might be used with advantage wherever the cost of the inoculation, by the experience of former years, does not exceed the losses caused by this disease. In 1895, however, the total losses after erysipelas inoculation amounted to 7½ per cent.! These astounding losses were attributed to vaccine matter of bad quality. In 1896, the loss represented 1¾ per cent.; in 1902, ¼ per cent. (234,000 inoculations).

In 1893, in *Würtemberg*, 10 out of 32 inoculated pigs died. The loss thus amounted to 30 per cent. The surviving pigs became more or less sickly. Further, the experiments made (on 117 pigs) in 1895–6 did not prove satisfactory.

In *France*, according to Chamberland, 1,067 died out of 75,455 young pigs which were inoculated during seven years (1886 to 1892); thus showing a mortality of 1.45 per cent. It is stated that before the introduction of the inoculation, the mortality amounted to 20 per cent., and in some districts even from 60 to 80 per cent. of all pigs.

In *Holland*, 1,829 pigs were inoculated during 1890, and 407 pigs during 1891; the result being that 1.7 per cent. of them died in consequence of the inoculation; 6.4 per cent. had their health more or less impaired, and the remainder experienced no ill effects.

2. **The Lorenz Method of Inoculation.**—In 1892 Lorenz published the results of experiments obtained from two inoculations, which were carried out as follows. In the first inoculation, a blood-serum obtained from artificially immunised animals (pigs, horses, sheep), and preserved by a ½ per cent. carbolic treatment, was used as injection (passive immunisation). In the second injection (3 to 5 days after), an erysipelas broth-culture was injected (active immunisation). The protection afforded by the inoculation lasts about half a year, and may be prolonged to a year by repetition of the second inoculation. The serum of many diseased animals, too (75 per cent.), possesses a curative influence. In Leclainche's procedure, again, both inoculations were carried out at one operation (simultaneous inoculation).

According to the experiments carried out up to the present, especially those in Prussia, Würtemberg and Hesse, the Lorenz

method has had very favourable results. In 1897–9, for example, 217,376 pigs were inoculated in Prussia (Prenzlauer vaccine); 69 per cent. of the pigs affected with erysipelas were cured by the inoculation. The total loss attributable to the inoculation was but just over ½ per cent. In 1899–1900, 50,000 pigs were inoculated with Landsberger serum, 30,000 of these being simultaneous inoculations, and, professedly, with the best results; the total loss reached only ¼ per cent. (½ per cent. of erysipelas inoculation). In 1901, 97,000 pigs were inoculated with the Landsberger serum, 77,000 of these being simultaneous inoculations; the losses were but 5 per 1,000; of the curative inoculations, 77 per cent. were favourable in result. In 1901, 163,000 pigs were inoculated with Prenzlauer serum: the losses due to inoculation were less than ¼ per cent.; 70 per cent. of the curative inoculations were successful. In Würtemberg there were, in 1899, 12,229 pigs inoculated; in 1900, 15,208; in 1901, 20,789; in 1902, 27,811—thus in the course of 4 years 75,000 pigs were treated, with the result of favourable protection in all the cases, and with no losses due to the inoculation.

3. **Susserin.**—An erysipelas serum, prepared in the Höchster dye works, was recommended in 1899 by Schütz and Voges. It was first employed by itself, as a protective and curative agent; but soon after, from the analogy of the Lorenz method, it was used in conjunction with erysipelas cultures. It was reported that for 1899, the results obtained with "susserin" were as favourable as those derived from the Lorenz serum. According to the reports for 1900, 49,000 pigs were inoculated with "susserin" in Germany (145,000 according to the Lorenz method); the losses due to inoculation were just under ¼ per cent. In Baden, in 1900, the number of successful curative inoculations with "susserin" was 83 per cent.

An erysipelas serum was also prepared in a manufacturing vaccine laboratory in Heilsberg in East Prussia. The susserin, and also the Landsberger and the Heilsberger erysipelas serum are subject to State control in the serum-testing institute at Frankfort-on-the-Main; the Prenzlauer serum to the control of the Hygienic Institute of the Berlin Veterinary College.

4. **Porkosan.**—Under this name there was recommended in 1896, a therapeutic remedy prepared from erysipelas cultures, and a simple injection was used for protection against erysipelas. After that the practical application of the remedy had frequently

shown, on the one hand, its inefficacy, and, on the other, the fact that it produced serious inoculation disorders and led to great losses; it was further established by the scientific investigations of Musehold, Johne, Voges, Schütz, etc., that "Porkosan" does not, generally speaking, produce an immunising effect.

Transmission of Swine Erysipelas to Man.—Transmission of the erysipelas of pigs to man has been observed by Casper, Jensen, Cremer, Mayer, Hildebrandt and Hartmann (slaughter, infection by inoculation-syringes). The local changes consist chiefly of an erysipelas of the seat of infection (finger, hand), with painful swelling and sometimes a formation of vesicles; in some cases, high fever and swelling of the neighbouring joints are observed. Recovery generally takes place after 8 to 14 days. Lubkowski found erysipelas bacilli in the intestine of a five-year-old child, who suffered from intestinal catarrh with vomiting and jaundice. Erysipelas infection of man from consumption of this pork has not been observed; the flesh of erysipelatous pigs is, accordingly, not to be regarded as unwholesome.

II.—CONTAGIOUS PNEUMONIA OF THE PIG AND SWINE FEVER.

Etiology and Pathogenesis.—(1) *Contagious pneumonia* of swine is caused by the bacillus suisepticus, which is identical with the ovoid bacterium of the German swine pneumonia, first discovered by Löffler and Schütz. Also with that of the American swine-plague, found by Salmon; and with the bacillus of the English and French pneumo-enteritis, which was cultivated by Klein, Galtier, and Chantemesse; as well as with the so-called vacuole-bacillus found by Bang in Danish pigs. The bacillus suisepticus belongs to the group of septicæmia hæmorrhagica or Pasteurellosis (septicæmia of rabbits, deer- and cattle-plague, swine pneumonia, fowl cholera). It is a bacterium of about 1 μ in length, and 0.5 μ in breadth; it varies in shape according to age, growth and environment; sometimes round, sometimes oval, or again in form of a short rod; non-motile, bipolar, staining with dyes, and on the other hand, not staining with the Gram re-agent, and not possessed of a flagellum. It is found in the diseased portions of the lung, and in the exudate of the serous cavities; in acute cases, also in the blood (septicæmia of swine). A similar bacillus is also present in healthy pigs, as a normal parasite, in the mucus of the mouth, throat and nose (so-called sputum-bacterium). The virulence of the bacillus suisepticus varies according to

its origin, the numerous genera and variations of the fungus, the environment, etc. (positive and negative inoculations; malignant, benign, hyperacute, acute and chronic course; pathogenic and non-pathogenic varieties).

Swine pneumonia is contagious in the highest degree. Many pig farms are real incubation foci for the production and distribution of the contagium. The points of entrance of the bacillus suisepticus into the body of the pig are not fully known. At all events, the lungs form, as a rule, in the usual aspects of the disease (affection of the lung), the place of entrance for the bacillus suisepticus. The demonstrative proof of this fact is offered by the positive occurrence of inoculation by inhalation, and by the intra-tracheal injection of the bacillus suisepticus in experimental pigs; as well as by the results of dissection, in which at the beginning, the lungs only—and usually, only a single bronchus is found diseased—forming the central point of the infection (Schütz, Karlinski, Joest, etc.). The infection is promoted by a catarrhal condition of the bronchi (chill, strongylus paradoxus). The lungs may also become secondarily infected from the intestine (swine fever). The intestinal infection is not, however, the rule in contagious pneumonia of swine, as Preisz considered (Joest). In the somewhat rare septicæmia variety of this disease, the points of infection are apparently in the outer skin (cutaneous and subcutaneous lesions caused by bites, whip lashings, etc.). Whether the bacilli normally found in the nasal and throat cavities of healthy pigs and similar in appearance to those of contagious swine-pneumonia, or the so-called "wild" swine-pneumonia bacteria, are by themselves alone, without the co-operation of other factors of contagious disease, pathologically efficient and capable of producing contagious swine-pneumonia, still remains undetermined. (Compare the analogous conditions of swine erysipelas.)

Pathogenic Effects of Bacillus Suisepticus upon Inoculated Animals.—1. Pigs are seldom, if ever, infected by cutaneous infection, but very easily by subcutaneous infection; and die, as a rule, from this cause. According to the virulence of the bacilli, various grave inoculation diseases are developed. Very virulent bacilli kill pigs after from 1–3 days (diffuse swelling around the site of inoculation, high fever; erysipelatous-hæmorrhagic œdema of the skin, subcutaneous tissue, and muscular strata; cloudy swelling of the parenchyma of the various parts of the body). Less virulent bacilli kill pigs after an interval of from 1 to 3 weeks (pronounced swelling at the seat of the inoculation, acute hepatitis, hepatic cirrhosis and jaundice; with partial hepatisation, gaseous necrotic foci and cavities in the lung). Pigs also die after intra-pulmonary

(necrotic pneumonia with sero-fibrinous pleuritis), intra-tracheal, intravenous, intraperitoneal and intrapleural infection, as well as after inhalation of bacilli. On the other hand, pigs may not sicken on being fed with the bacillus suisepticus—carried out with a view to intestinal infection (Schütz, Salmon, Priesz, Prettner, and others).

2. In horses, subcutaneous injection produces a very marked œdema at the seat of inoculation; intravenous injection, the appearance of an acute or chronic intoxication (high fever, metastatic tendo-vaginitis and arthritis, paralysis of peripheral nerves, thrombosis, lumbar weakness, and cirrhosis of the liver, similar to that which is known in Germany as "Schweinsberger" sickness).

3. Cattle show the same symptoms after subcutaneous and intravenous injection as horses; so do goats and sheep.

4. Doves and hens are less susceptible; it is only by large doses of very virulent bacilli that they die after an average interval of from 1 to 8 days.

5. Guinea-pigs die from subcutaneous injection after from 2 to 3 days; after intraperitoneal injection even after from 9 to 18 hours; they are not infected cutaneously. Rats are but slightly susceptible.

6. Rabbits are very susceptible; they perish after every mode of introduction into the body (also by the results of cutaneous injection and of feeding) with the phenomena of septicæmia of rabbits, peritonitis and pleuritis; pronounced hæmorrhagic inflammatory œdema at the seat of inoculation.

7. Mice are among the most susceptible of all; they die from cutaneous and subcutaneous inoculation of virulent bacilli after an interval of from 24 to 48 hours (inoculation-œdema, septicæmia); after feeding, within 3 days.

2. **Swine Fever** is produced by the bacillus suipestifer (swine fever bacillus). This parasite is identical with that discovered by Salmon in 1885, and first described satisfactorily by Salmon and Smith, the American cholera-bacillus (hog-cholera); also with the Danish fever-bacillus of Bang and Selander (Danish swine-plague or swine-diphtheria). This bacillus forms short rods, 1.2–1.5 μ long, and 0.6 μ broad, with rounded ends; and is characterised by lively movement, and the possession of flagella; while it is easily stained in an aqueous anilin solution. It is also stained by Gram's method. Spore-formation does not occur. It is most readily found in the mesenteric glands and spleen in an acute case of the disease; in the chronic form, it is found most readily in the mesenteric glands, though its existence is often difficult or even impossible to prove. Besides, it varies in form and virulence; and in the cadaver is often associated with other bacteria, and especially with the bacillus suisepticus, so that its bacteriological determination is often difficult.

With regard to its biological relations, experiment has shown

that the bacillus suipestifer is killed in 15 minutes by a temperature of 140° F. Its resistance to cold is, on the other hand, considerable ; for instance, it can survive for a month at a temperature of 42.8° F. In the upper strata of the earth, and in ordinary drinking water, it retains its virulence for about 4 months. It is rapidly killed by formaldehyde, freshly-burned lime, and milk of lime (1 per cent.) ; in concentrated solutions of common salt, on the other hand, it dies only after 4 weeks (Salmon and Smith).

The infection of pigs by these bacilli is easily effected through the alimentary canal, as feeding experiments have proved. They produce, according to their virulence, either an acute hæmorrhagic enteritis, with grave general infection ; or chronic inflammatory processes in the intestine (Jensen). Frequently, a mixed infection takes place: with the bacillus suisepticus (Preisz), and with the necrosis bacillus (Bang). A secondary contagious swine-pneumonia infection is especially prone to occur, according to Preisz, where intestinal lesions have developed in the course of swine fever ; when pigs often die very rapidly, in many cases, of pleuro-pneumonia ; even before the swine fever has produced morbid changes in the body (septicæmic swine fever).

Swine fever is extremely contagious. Infection occurs either directly from affected pigs, especially from the excrement, or indirectly, through various media ; above all, through the attendants, the food, and the feeding-troughs (the distribution of the disease by carrion crows has also been noticed by Morton). According to the researches of Theobald Smith, chronically affected and thoroughly diseased swine are latent sources of infection ; as from them an output of pathogenic bacilli continues to take place long after the subsidence of the manifest phenomena of the disease. Karlinski states that the bacillus suipestifer is found in the dung of artificially infected pigs from 6 days onwards, and is discoverable up to the fortieth day. It displays in swine-sheds, also, a great power of resistance to natural and artificial disinfecting agents (hence the difficulty of disinfection !). Immunity generally remains after an attack of swine fever (see Chapter on Inoculation).

The causes of the distribution and dissemination of both these plagues are to be looked for in the management of pigs. The pig markets, the hawking from house to house, and the recourse to boars of other breeds, furnish the most fruitful sources of the disorder. As both diseases possess relatively long periods of incubation, and the contagium, by virtue of its great

resisting power, remains active for many weeks in the dealers' sheds and in the cattle trucks, the disease is very often conveyed by apparently quite healthy animals. Various other agents also serve as media (clothes, men, healthy pigs). Predisposing influences are chills in transport, sudden change of food, and great fatty deposit (Rust).

The incubation stage of contagious swine-pneumonia and swine-fever are considerably longer than that of erysipelas. It lasts, on an average, from 8 to 14 days, and at least 5 to 10 days.

Effects of the Bacillus Suipestifer on Inoculated Animals. —1. Pigs are very susceptible to the fever bacillus. The infection takes place most easily from the intestine, which is contrary to contagious swine-pneumonia. Feeding with viscera infected with the bacillus is much more effective in this way than that with pure cultures (influence of the gastric secretion). The intestinal changes (ulcers and necrosis in the large intestine) correspond, in the inoculation by food, exactly with those of the natural disease. The localisation of the disease in the large intestine is produced by the prolonged retention of the intestinal contents, and the multiplication of the bacteria which is favoured thereby. The feeding with the viscera succeeds with 90 per cent. of all the animals experimented on ; and kills them, as a rule, within a period of 7 to 21 days, with the typical symptoms of swine fever. Subcutaneous injection succeeds with greater difficulty (only with a high degree of virulence and in large doses) ; death occurs after from 7 to 18 days. The local inoculation swelling is much smaller than in cases of contagious swine-pneumonia (only about the size of a hen's egg) ; the intestinal lesions are those of swine fever (swelling, necrosis and ulceration of the follicles in the large intestine, sometimes, also, nodules in the kidneys of the size of a hazel-nut). In a rapidly progressive case, it develops, as in cases of intravenous injection, the appearances of a hæmorrhagic septicæmia. The disease may also be developed from the lungs, from intra-pulmonary injection (swine-fever pneumonia).

2. Horses, cattle and sheep are not killed by subcutaneous inoculation of swine-fever bacilli, abscesses only being formed.

3. Doves die in from 1 to 3 days after subcutaneous and intra-muscular infection.

4. Rabbits are very susceptible, and thus form the most favourable subjects for experiments. They die after cutaneous and subcutaneous infection in the course of from 3 to 14 days, and show a typical liver disease, in the form of numerous whitish-yellow nodules on the surface (multiple coagulation-necrosis, the result of capillary obstruction by the bacteria). Intra-pulmonary injection develops necrotic pulmonary inflammation and intestinal infection, and after feeding (with the bacteria) a typical follicular necrosis of the intestine, with formation of ulcers. Guinea-pigs are similarly affected (death occurring after subcutaneous injection in about 4 days).

5. Mice are very susceptible, and die within from 3 to 7 days after subcutaneous infection (multiple hepatic necrosis) ; and they also rapidly succumb after inoculation by feeding.

Anatomical Conditions.—*Pure contagious pneumonia of the pig* (bacillus suisepticus) is characterised, in acute cases, anatomically by a pneumonia with formation of multiple necroses, a hæmorrhagic fibrinous pleuritis and pericarditis; also by general septicæmic changes (swelling of the lymph-glands, hæmorrhages in the kidneys and other organs); and in the chronic form, by catarrhal pneumonia of the anterior lobes.

Pure swine-fever (bacillus suipestifer) is distinguished by intestinal inflammation and ulceration of the croupous and diphtheritic type (caseous button-like plaques in the large and small intestine, especially at the ileo-cœcal valves); swollen, hard, necrotic glands in the mesentery; and, under certain circumstances, local necroses in the skin and kidneys.

In many cases, however, contagious swine pneumonia and swine fever cannot be anatomically distinguished (Preisz). Both diseases may be accompanied by hæmorrhages, croupous gastro-enteritis, pleuritis, pericarditis and peritonitis. In addition, we have the fact that often both diseases are present at the same time (septicæmic swine fever). Finally, the anatomical changes vary according to the virulence of the infective matter, the resisting power of the individual animals and different breeds of pigs, as well as according to the course of the disease, and the localisation of the morbid processes.

CONTAGIOUS SWINE PNEUMONIA.

1. *The lung*, in acute cases, presents an appearance of multiple necrotic pneumonia. On section, dark-red or grey-red, hepatised spots of different sizes, particularly in the anterior lobes and at the root of the lungs, are found. These spots show on their cloudy, slightly granulated section surface, numerous yellow or grey-yellow (buff-coloured), dry, caseous, circumscribed foci, which, in peracute cases, are punctiform and about the size of grains of fine oatmeal. In acute and chronic cases they vary in size from a pin's head to a hen's egg, or may be even larger, and are then sharply defined from the other lung tissue by a red, inflammatory line of demarcation, and later on by a firm capsule of connective tissue. These yellow deposits are dead portions of lung tissue formed in a manner exactly similar to that which takes place in equine contagious pleuro-pneumonia, in consequence of a multiple inflam-

mation of the lungs with hæmorrhagic exudation and subsequent discoloration. A large area of necrotic lung tissue may be formed by the running-together of several of these foci. In some cases no necrotic foci are present, and then there is only a diffuse hæmorrhagic pneumonia. The larynx, trachea, and bronchi contain a frothy, serous, or sanious fluid. The bronchial glands and the mediastinal glands are greatly swollen, reddened and infiltrated. They are also frequently hæmorrhagically infiltrated, and their capsule is filled with blood. In the nasal cavities we frequently find punctiform hæmorrhages (hyperacute form) as well as acute and chronic nasal catarrh. In chronic contagious swine-pneumonia, the alterations are confined to a catarrhal inflammation of the anterior lobes, which is sometimes associated with a fibrinous pericarditis.

2. The *pleuræ*, especially in cases of gangrenous pneumonia, sometimes show signs of acute, serous, or sero-fibrinous pleuritis; and in cases of long standing, signs of chronic adhesive pleuritis. In the former case, the more or less copious exudate is either clear and watery, or is opaque, mixed with flakes of fibrin, and is often found on both sides. Its extensive accumulation may lead to hydro-thorax with compression of the lungs. Under the coating of fibrin, the pleuræ appear cloudy, lustreless, and raised, like velvet. Adhesion of the visceral and parietal layers of the pleuræ frequently takes place as a consequence of chronic pleuritis, when we often find that almost the entire surface of the lungs has become adherent to and united with the costal pleuræ.

3. The *pericardium* generally contains a considerable quantity of opaque or bloody fluid. In some epizoötics, a fibrinous pericarditis, which occasionally leads to adhesion of the pericardium, constantly occurs. The muscular substance of the heart is relaxed, and is of a yellowish brown colour; in acute cases, hæmorrhages are found in the substance of the auricles and of the papillary muscles.

Variations in Contagious Swine-Pneumonia.—Ostertag states that this disease has of late prevailed in the less virulent chronic form, while its character has materially changed in North Germany during the past twenty years. Until about from 1894 to 1898 the plague appeared essentially as acute, croupous, necrotic pneumonia, with fibrinous pleuritis, pericarditis, and, in isolated cases also, as peritonitis. Since then it has manifested itself principally in the form of chronic catarrhal pneumonia (greyish-red, atonic hepatitis) and is limited to the outside parts of the lung (mainly the fore lobes). Even now, acute, as well as chronic cases occur, when chronically diseased animals are exposed to the ill effects of trans-

port, or when the disease has been carried by affected pigs into clean sties. The infectious catarrhal inflammation observed by Greve in pigs in Oldenburg, has been placed by Ostertag in the same category. The bacillus pyogenes suis, which Grips held to be the origin of contagious swine-pneumonia, plays, according to Ostertag, only a secondary part in the infection (the cause of ulceration and caseification).

II.—SWINE FEVER.

1. In peracute and acute cases, the *mucous membrane of the stomach and intestines* frequently show signs of hæmorrhagic gastro-enteritis, such as, great congestion, swelling, the presence of ecchymoses and ulcers, and the fact that the fluid contents of the intestines are thin and sanious. The intestinal changes are very characteristic, and consist essentially in a necrotic, caseous, intestinal inflammation, which specially involves Peyer's patches. A very characteristic *post-mortem* appearance is the presence of more or less numerous yellow or grey-yellow [buff-coloured], cloudy, circumscribed spots or nodules in the mucous membrane of the posterior part of the small intestine, cæcum, colon, and especially on the seat of Peyer's patches. These spots and nodules are somewhat prominent and are of different sizes. At other times, they are superficial and are only as big as the head of a pin or a lentil ; or they take the form of dry, round nodules that are about the size of a pea or a button, and extend through the entire intestinal wall as far as the serosa, which shows circumscribed changes due to peritonitis. Besides these nodules, we find on the intestinal mucous membrane, numerous buff-coloured, cloudy spots (commencing diphtheritis), or ulcers which are of different sizes, depth and form, and which sometimes reach to the serosa.* Finally, there has also been observed a hæmorrhagic, as well as a croupous and diphtheritic, inflammation of the mucous membrane of the mouth (tongue), pharynx, and œsophagus. The mesenteric glands and the portal lymph glands are swollen and hæmorrhagically infiltrated, and frequently undergo caseation in chronic cases. The liver is enlarged and its edges are rounded (parenchymatous

* McFadyean states that in naturally occurring cases of swine fever, diffuse diphtheritic inflammation (superficial necrosis), or, more frequently, ulcers are to be found, with few exceptions, in the large intestine *post-mortem* ; and that in the very rare cases in which these lesions are absent, we may observe in their place intense congestion and punctiform hæmorrhages.—Tr.

and acute hæmorrhagic hepatitis; multiple necrotic foci, and further severe inflammation of the gall-ducts and gall-bladder). The spleen, on the contrary, is, as a rule, more or less normal in size and condition in acute cases. In chronic cases, caseous foci are sometimes found in it.

2. The *peritoneum* frequently shows signs of having suffered from a fibrinous inflammation causing cloudy swelling, congestion, hæmorrhagic infiltration, and the formation of a velvety coating of fibrin on the surface of the peritoneum, and the effusion of a considerable quantity of yellow exudate mixed with flakes of fibrin. In chronic cases, we find adhesive peritonitis with adhesion of the intestines, especially of the intestinal loops. Hæmorrhagic nephritis is sometimes present with pyelitis and cystitis.

In the peracute form, we sometimes find only general changes of septicæmia, without any particular local affection.

In old standing cases of either disease, the dead body is greatly emaciated and very anæmic. In the muscles, we find a parenchymatous and hæmorrhagic myositis. The cutaneous muscle is sometimes in a necrotic state. The skin displays a diffuse erythema; hæmorrhages; also circumscribed and diffuse necrosis. The skin is covered with firm crusts, the removal of which displays ulcers or commencing cicatrisation.

Symptoms.—Pure *contagious swine-pneumonia* appears under the form of an infectious pneumonia, a cough being the chief symptom. We can distinguish an acute or chronic pectoral type, and a more rare, peracute, septicæmic variety. Pure *swine-fever* takes the form of an intestinal inflammation of epidemic course, the most important symptom of the disorder being diarrhœa. We can distinguish the usual chronic intestinal form, and the peracute septicæmic, hæmorrhagic one. Both diseases can appear, in their pure forms, as distinct epidemics, more usually, however, they complicate one another; not only among a certain group of pigs, but even in an individual case. A clinical delineation of individual cases is, therefore, difficult. For this reason, both forms of disease are clinically described together in the following pages. For practical purposes it would seem to us better to divide it clinically into an acute, a chronic, and a peracute form.

1. The *acute form* appears as a pneumo-intestinal inflammation which is sometimes complicated with various

kinds of exanthemata of the skin. The duration of this form of the disease, which is the one most frequently observed, varies from a few days to 2 or 3 weeks. There is a moderate fever (from 103° to 105° F.); short, dry, painful, and spasmodic cough, often lasting for a quarter of an hour, with attacks of suffocation; muco-purulent discharge from the nose; heavy pumping and panting breathing; cyanosis of the mucous membranes of the head; and dull percussion sound of the thoracic walls. Auscultation proves the existence of rhonchi and suppressed respiratory murmurs. The animal, which at first is sometimes more or less excited, soon manifests great depression and weakness, and loss of appetite; and may have difficulty in swallowing solid food. Constipation is generally present at the beginning of the disease, and often changes, later on, into diarrhœa, which may, however, be present from the commencement, if the digestive apparatus is the chief seat of the disease. In grave cases, diphtheritic ulcers form on the tongue, gums, buccal membrane, palate, and tonsils. The eyes are sunken, and there is sometimes conjunctivitis, keratitis, discharge from the eyes, and swelling and adhesion of the eyelids. In many cases the skin shows no striking changes; but in others we may find diffuse or spotted redness, vesicles and wheals with violent pruritus, and finally crusts which frequently cover the whole body, and which vary in size from a pea to a sixpence. According to Schindelka, three different forms of cutaneous changes occur in contagious swine-pneumonia, namely: (*a*) A spotted exanthema on the region of the anus, lower surface of the body, and inner and outer surface of the thighs, which lasts only for 3 or 4 days, and takes the form of red spots the size of a lentil. Vesicles, and finally circular, brown-yellow crusts appear on these spots. (*b*) A pink uniform exanthema which is not sharply defined and shows a red rich discoloration of the skin. It begins at the anus and spreads in 3 days over the lower surface of the body. It becomes pale from the fourth day, on account of the epidermis desquamating in small scabs. (*c*) An attack of urticaria with the formation of vesicles (urticaria bullosa), in which case there is an eruption of very rapidly crowding wheals, upon which vesicles, pustules, and crusts quickly form.

2. The *chronic form* of contagious pneumonia of the pig develops from the acute form, which we have just described. Its course lasts on an average from 4 to 8 weeks; but may some-

times be prolonged for several months. According to its localisation, the symptoms consist either of a chronic affection of the lungs, or of the intestines. The former appears as a chronic cough without further marked symptoms, or as a pulmonary consumption with persistent dyspnœa, chronic cough, and great and continually increasing emaciation. The latter manifests itself particularly by the presence of fœtid, green or greenish-yellow diarrhœa, which lasts until the animal dies, or which may alternate with constipation. The patient loses condition, especially about the hind-quarters; becomes anæmic; and shows great weakness and symptoms of paralysis. Palpation of the abdomen is painful, and nodular tumours are sometimes felt in it. In both these forms of the disease, the skin is frequently covered to a considerable extent with scabs, and the eyelids are closed up by crusts. Pulmonary and intestinal symptoms are often simultaneously present in the same animal.

3. The *peracute form* (Rust and Schindelka) of contagious pneumonia of the pig is so severe—death ensuing in from 3 to 10 hours—that it can seldom be studied. It consists of a highly feverish hæmorrhagic inflammation of the lungs, or of pure septicæmia. The animal suddenly becomes extremely ill, after it has been feeding with a good appetite only a short time previously. It suffers from rigors, staggers, and falls down; the temperature in the rectum rises to from 104° to 107° F.; the pulse is very frequent and irregular; the beating of the heart is almost imperceptible; respiration is difficult and accelerated; tracheal râles are present; there is a frequent moist cough; the expired air is very warm; the upper surface of the body feels as hot as fire to the touch; the sufferer, when taken hold of, offers but little resistance; the grunting is faint and muffled; and we may sometimes observe a mucous discharge and hæmorrhage from the nose, and red spots on the skin about the ears, on the neck, and on the sides of the chest. The posterior part of the body is somewhat tympanitic. Pressure on the region of the stomach causes pain, and the animal sometimes shows an inclination to vomit. Death often supervenes in the course of a few hours (on an average from 3 to 10); and sometimes the peracute form passes into the acute.

The *course* is unfavourable in all three forms. That of the peracute type is, as a rule, fatal. In the acute form, recovery is rare; it either ends in death or passes into the mostly incurable chronic variety

Prognosis.—Contagious swine-pneumonia and swine-fever are formidable animal plagues, which prove destructive to the breeding of pigs, and even to the national wealth in many countries. The proportional mortality in the acute forms reaches an average of 70 to 80 per cent. As with many other disorders, there are observed a milder and sometimes a more malignant type. Besides, contagious swine-pneumonia sometimes alternates with swine-fever. The complication of swine-fever with contagious swine-pneumonia involves a specially malignant course. Swine-fever is a particularly destructive disease for young pigs, up to the age of four months. Chronic contagious swine-pneumonia produces fewer deaths; but there is a great commercial loss in consequence of the impaired fattening of the infected animals. With regard to the losses in various countries, the following facts are known:

1. In North America contagious swine-pneumonia and swine-fever first, nominally, appeared in the middle of the 19th century. A general distribution of it took place in the years 1873 and 1878. The loss then reached 75 per cent. In 1885, the total losses in the United States represented over £6,000,000; in Missouri alone, 200,000 died; in Nebraska and Indiana, 400,000 (one-fourth of the whole stock). In the state of Indiana the losses in the year 1887–1897 amounted to £600,000. In the State of Iowa, the yearly loss was estimated at over £3,000,000.

2. These diseases first attained a wide distribution in England in 1862; the mortality reached 75 per cent. In 1885, 40,000 pigs sickened; of these, 27,000 were slaughtered, and 10,000 died. In 1886, 35,000 died.

3. In Austria-Hungary, the losses in 1895 amounted to £2.500,000; and in 1896, £2,900,000. In Hungary alone, in 1895, 413,562 pigs were affected; in 1896, 868,777; in 1897, 514,291; in 1898, 318,000; in 1902, 227,000. The mortality, in 1895, reached 81 per cent; in 1896, 74 per cent.; in 1897 and 1898, 70 per cent.; in 1899 and 1900, 60 per cent.; in 1902, 57 per cent.

4. In Germany, in 1897, 11,400 cases of disease, with a loss of 78 per cent., were officially notified; the provinces of Breslau, Liegnitz, Oppeln, Posen and Trier were more especially infected. In 1898, 11,800 pigs were affected (with a loss of 81 per cent.); in 1899, 12,000 (with 82 per cent. loss); in 1900, 18,000 (85 per cent. loss); in 1901, 38,000 (81 per cent. loss); and in 1902, 45,000 (with 78 per cent. loss).

Differential Diagnosis.—Contagious swine-pneumonia may be confounded with various forms of sickness ; the doubtful cases being decided by bacteriological investigation. The following are the chief diseases with which it may be confused.

1. *Epizootic erysipelas* may be easily mistaken for the eruptive form of contagious swine-pneumonia, which may assume even the appearance of urticaria. The bacteriological and anatomical result will decide the question, also inoculation (in contrast to erysipelas, swine pneumonia is not fatal to pigeons).

2. *Tuberculosis.* The chronic necrotic form of contagious pneumonia and swine fever possesses great similarity to tuberculosis ; because it also gives rise to caseous foci in the lungs and in the intestines. Apart from the difference in the respective bacteriological results, we have the fact that tuberculous deposits become calcareous in time, and are, also, of various ages, as a rule. Tuberculosis, besides, is frequently generalised ; and tubercles are found in the neighbourhood of the tubercular ulcers.

3. *Phthisis verminalis* has been confused in former times with contagious pneumonia of the pig, in cases in which pulmonary worms were found in the lungs of pigs suffering from this disease.

4. The distinction of contagious swine-pneumonia and swine-fever is often difficult ; the doubtful cases must be decided by bacteriological examination and inoculation. The sero-diagnostic investigation is not reliable.

Therapeutics.—Medicines, as a rule, are of no use. The intravenous injections of sublimate, which have recently been recommended, have not proved satisfactory. *Prophylaxis* is all important in these diseases, which can be successfully combated only by inoculation and stringent veterinary police measures.

In Germany, the procedures were formulated in the proclamation of the State Chancellor, September 8th, 1898. And, further, in the modification and generalised application of the ordinance relating to cattle plague, the police regulations for the slaughter of diseased and suspected pigs were brought into prominence. There are also in most parts of Prussia, police regulations applied to the measures to be taken against both these diseases, which agree, for the most part, with the draft given below. Of the various vaccines which have been recommended in this connection, the " polyvalent " serum of Wasser-

mann and Ostertag, obtained from various sources of the diseases, has proved specially satisfactory (see below).

The period of warranty, according to the German Civil law, is ten days—for both diseases.

The statements which Zschokke made at the International Veterinary Congress at Berne in 1895 essentially agree with our theory of the identity of contagious pneumonia of the pig and swine fever. He states that it is hardly possible to find any difference between the respective bacteria, which evidently belong to the same group and differ morphologically only on account of differences in external influences (quantitative, not qualitative, difference). In any case, the two diseases cannot be separated pathologically, anatomically, or from a veterinary police point of view. Zschokke drew the following conclusion, which was almost unanimously accepted by the Congress: "The two diseases (contagious pneumonia of the pig and swine fever), from etiological and practical reasons, should, under a common name, be added to the diseases which ought to be combated by the State; their compulsory notification should be enforced; and they should be included in the catalogue of epizoötics, and should be separated, in it, from bacillar erysipelas."

[In England the law does not take any account of swine-pneumonia or swine-erysipelas, because these diseases seldom or never occur in the epizoötic form in Great Britain.

The Departmental Committee appointed in 1895 recognised the unity of swine fever, which they say "cannot arise under any conditions which exclude the specific virus; in other words, it is not a sporadic disease, but one of the true contagia." The Committee did not extend their experimental researches to swine erysipelas, which they "believe to be of rare occurrence in this country in the epizoötic form. A considerable number of cases of the chronic form, with valvular disease of the heart, have been discovered during the *post-mortem* examinations conducted by the veterinary officers of the Board. But when it has been possible to trace the history of the pigs from which the diseased hearts were taken, it has been ascertained that the affection had not manifested any tendency to spread by contagion."

The following extracts from the Report may be of interest:—

"Swine fever may be defined as a contagious and infectious disease of the pig associated with a necrotic and ulcerative condition of the mucous membrane of the intestine, the morbid condition being nearly always most marked in the large intestine.

"The disease of the lungs which occasionally accompanies the disease in the intestine is either collapse or pneumonia. It is necessary, however, to observe that in none of the experiments performed for the Committee was pneumonia produced either by inoculation with pure cultivations of the micro-organism, or by feeding with the natural material obtained from animals suffering from swine fever.

"In very acute cases of swine fever, the disease may prove fatal within 2 or 3 days, and then the only lesions present may be intense inflammation of the stomach or intestines, or of both; and in these exceptional instances a certain diagnosis cannot be made except

by bacteriological methods. . . . The acute and rapidly progressive form of the disease is commonly met with. There are also, and always have been, many cases of the obscure or chronic form of the disease, in which the morbid changes go on slowly for many weeks or months, and finally attain an excessive state of development without being attended by any symptoms which are usually accepted as diagnostic of swine fever.

"Some very important information in regard to the obscure form of swine fever was obtained by the Committee from the examination of swine which had been isolated for a period of 2 months on infected premises. At the end of that time they had been certified by a veterinary surgeon to be free from swine fever, and would in the ordinary course have been released. In several of these instances, instead of being released, the swine were, at the request of the Committee, slaughtered, and the organs sent for examination. In each set of specimens, characteristic lesions of swine fever were detected.

"It was found that animals placed in contact with the diseased swine, or in the sties which had been occupied by them, became affected with a similar type of slowly progressive disease. On *post-mortem* examination of all the original cases, the remarkable feature was the great disproportion between the very advanced lesions in the digestive canal, and the slight symptoms of disease exhibited by the animals during life."

Referring to clinical observations on the progress of swine fever among pigs which had been infected by feeding, by inoculation, or by exposure to natural infection, the Committee state :—"It will be noted that, taking the whole of the results, they were remarkable and quite unexpected. The well-known and characteristic symptoms of swine fever were not developed at any time during the course of the disease, which, in some cases, assumed a very exaggerated form. There was no redness of skin apparent in any case, and in one instance only were there any indications of loss of power in the hind extremities. Diarrhœa occurred in a few cases and continued for a short time, but in other instances constipation was present for a few days. The appetite was variable in some of the animals, but altogether they consumed a fair quantity of food up to the time of death or slaughter. In nearly all the swine which were examined *post-mortem*, very pronounced lesions of swine fever were detected. In some cases there were large intestinal ulcers partly or entirely healed, the animals having, in fact, recovered from the attack."

Alluding to the chronic form of swine fever in four pigs, which while in one pen, had been fed on the intestines of a pig which had suffered from this form, the Committee observed that "none of the ordinary symptoms of acute swine fever, *i.e.*, discoloration of skin, loss of motile power in the hind extremities, cough, and diarrhœa, appeared, but instead the indications which are now unhesitatingly accepted as evidence of the chronic or obscure form of the disease, were developed early in the experiment. After a few days the eyes became dull and slightly sunken, the upper lids had a peculiar appearance due to the loss of hair, exposing the skin, which was covered with desquamating cuticle. A sticky secretion was discharged from the edges of the lids, the skin of the forehead was wrinkled, the hair (bristles) was slightly

elevated over the surface of the body, the tail was limp and often the tips of the ears were dropped forward, and the animal exhibited a generally dejected appearance. Constipation alternated with diarrhœa from time to time, but neither symptom was so marked as to excite special attention. The pigs ate a good quantity of food, but never improved in size or in condition; indeed the tendency in these animals, as in the other cases recorded, was in the contrary direction.

"The pigs usually attacked their rations with avidity when first placed before them, but often ceased to eat before finishing their meal, returning to the trough again after an interval of rest.

"On January 30th 1 of the 4 pigs was killed, and on *post-mortem* examination well defined lesions of swine fever were detected. There were necrotic ulcers on the ileo-cæcal valve, and several large ulcers with necrotic centres in the colon, and the mesenteric glands were as usual highly congested. Other organs of the body showed no indications of disease.

"It was deemed expedient to test the remaining 3 pigs as to their power to transmit the disease by association, and accordingly 2 young pigs were put in the pen with them on February 5th.

"On February 14th both pigs presented the appearance which has lately come to be recognised as the 'swine fever expression.'

"With very slight and temporary alteration of symptoms the 2 lately introduced pigs and the 3 which had been fed on December 24th remained until February 22nd, when they were all slaughtered.

"On *post-mortem* examination of the 3 fed pigs, lesions were detected very similar in character and extent to those which were found in the pig killed on January 30th, but in all of them the majority of the ulcers were undergoing the healing process, and the mucous membrane in several parts of the cæcum and colon showed quite complete cicatrisation. It was evident that the animals were recovering.

"The lesions of swine fever in the 2 pigs which had been kept in contact with the 3 pigs fed since February 5th were well defined, but the ulcers were in one case very small and numerous in the colon, and most of them in both pigs were healing rapidly, and there is no doubt that the pigs would have recovered."—TR.]

Veterinary Police Regulations.—The Royal Technical Commission for Veterinary Regulations in Prussia, promulgated the following rules in *1896, for the arrest of contagious swine-pneumonia and swine-fever.* These are, as compared with those of erysipelas (see Vol. II., p. 238), considerably more stringent.

1. Duty of notification in cases of outbreak, and no suspicion of the disease.

2. When a pig is found to be suspiciously affected, all the animals associated therewith, and which have come into contact with it, are to be kept shut up under observation till the official veterinary surgeon has determined, as the result of careful investigation, if the suspicion was groundless.

3. When the outbreak of swine disease has been confirmed by the official veterinary surgeon, all the pigs in the infected farmyard are to be regarded as suspected of infection. The removal of pigs therefrom, or the introduction of others thereinto, must not be attempted without

police supervision. For purposes of immediate slaughter, or enclosure in isolated farm-yards, the removal of suspected swine must be carried out in well-closed waggons. The closure of the affected farms for at least six months is to be enforced.

4. When more than one farm-yard in a locality is infected, the complete or partial closure of the infected neighbourhood against the removal and the transit of pigs must be carried out by the police. But healthy pigs destined for immediate slaughter may be conveyed out of the infected locality in waggons or by the railway.

5. All droves and other means of transport of pigs, in a locality in which the outbreak of the disease has been established, must be restricted for at least six months. When the pigs suspected of infection have been at once slaughtered by direction of the owner, their subsequent conveyance must be carried out with the provision that they are not to be brought into contact with healthy pigs during transport.

6. The cattle-markets are a source of great danger, and, therefore, the bringing of pigs to or from open markets in the affected localities should be forbidden.

7. The bodies of affected pigs which have died or been slaughtered, including the offal of the same, should be carefully destroyed.

8. The disinfection is to be carried out as in the case of erysipelas.

9. The protective regulations may again be rescinded when the whole of the affected herd has been removed by death or by slaughter; or when, on the infection of suspected pigs, no new case of sickness has arisen for at least six months after the last one; and when disinfection has been carried out.

In Austria, in the year 1899, a law was introduced for protection against swine-fever and the extinction of the disease. It provided that all pigs affected with symptoms suspicious of the disease, and those suspected of exposure to infection, were to be slaughtered; and 95 per cent. of their value was to be refunded to the owners from the State Treasury.

Inoculation.—The inoculations which have hitherto been adopted are as follows:

1. In Italy, Perroncito and Bruschettini have recommended a vaccine prepared from contagious swine-pneumonia bacteria against both swine-pneumonia and swine-fever which has proved completely ineffective in the course of investigations carried out in Germany (Malkmus, Schlegel, Casper, Willach, Ostertag, and others); in Hungary (Ujhelyi, Urban, Fuchs, and others); and Italy (Gerosa, and others).

2. In North America, protective inoculations with old cultures were first made by Dettmers and Billings, but without result. According to Salmon they were productive of great losses and a wide distribution of the disease. Smith and Moore produced a form of the disease of chronic duration, of a period of months. Serum inoculations appear to have been more favourable. Peters, especially, has imitated the serum-treatment of erysipelas, injecting serum (170 mins.) (10 c.cm.) in combination with virulent swine-fever bacilli (17 mins.) (1 c.cm.). In 1897, 1,176 pigs were inoculated in this way; and, according to the report, 56 per cent. of the cases were successful. Schweinitz has also, of late, pronounced in favour of the serum-inoculation. He states that the best results are yielded by a serum which is a combination of both—that of contagious swine pneu-

monia with that of swine-fever. In the State of Iowa, in 1898, only 23 per cent. died, out of 1,727 pigs, which had been inoculated with serum; while of 3,197 which had not been inoculated, 81 per cent. were lost.

3. In Hungary it was often observed that pigs retained a state of immunity after a single attack of swine-pneumonia or swine-fever. Preisz inoculated 30 sucklings with the serum of a pig which had passed through the infection (170 mins.) (10 c.cm.); and then placed them with 30 which had not been inoculated, in contact with diseased pigs. Of the non-inoculated, all became infected and died; of the inoculated, 18 sickened, and 9 died. In 1897, 9,000 pigs were inoculated with serum; the greater number of these remained free from the disorder; while of those which contracted it only a few died.

4. In Germany, Kitt and Mayer found that the serum of horses which had been subjected to intravenous inoculation with fowl-cholera, on the one hand distinctly retarded the onset of contagious swine-pneumonia; and, on the other hand, displayed a life-preserving influence on the animals of experiment (rabbits and mice). Höflich inoculated 10 sucklings with the serum of a boar which had been infected with swine-fever and all remained free from infection. Ostertag found in his researches that pigs receive an active immunity against swine-fever from treatment with dead swine-fever cultures; but that, nevertheless, the blood-serum of pigs which have recovered from swine-fever is unfitted to procure immunity. A blood-serum with immunising qualities, may, on the other hand, be obtained by proper treatment of pigs, horses, cattle, sheep, and goats, with swine-fever cultures. Beck has, by immunisation of larger animals with virulent swine-pneumonia cultures, obtained a serum which is said to protect pigs against infection with swine-pneumonia, and to promptly cure those affected by the disease · the application of the serum is said to be without danger. It was also concluded by Beck that on giving it up, the disease increased. Müller used Beck's serum, with success, on 46 pigs. Schreiber made two preparations, from serum of animals that were immunised against swine-pneumonia and also against swine-fever, which protect healthy swine against both these diseases (protective serum), and cure animals affected with them (curative serum). The protective serum affords an immunity which lasts for one to five months. In 1900, 4,000 pigs were inoculated with Landsberger serum (septicidin), and 90 per cent. of these were protected. In 1901, 6,000 were inoculated; the protective inoculations were always followed by the desired results, when carried out in the prescribed form (serum-inoculation, subsequent inoculation with pure cultures); and the curative inoculations were successful in 64 per cent. of the cases. According to Schreiber, pigs remain immune to swine-fever after having passed through an attack of swine-pneumonia; on the other hand, those which have suffered from swine-fever present but an immunity of short duration against swine-pneumonia. In other places, unfavourable results have followed from septicidin inoculation (Höflich, Dammann, Kleinpaul, and numerous other observers). The Prussian official veterinary report for 1901, stated that the overwhelming majority of their correspondents had had unfavourable experiences of septicidin-inoculation. From the experiments of Höflich, septicidin has in no way corresponded to its representations; accordingly, he warns us against the secondary culture-injection, as the septicidin has not in a single case afforded protection.

The results hitherto obtained in the campaign against swine-pneumonia

with the aid of the **polyvalent serum**, according to Wassermann and Ostertag, are more favourable. Up to 1903, 11,000 pigs (9,000 of these sucklings under three months old), had been inoculated in 253 localities. The result of inoculation being satisfactory: 85 to 88 per cent. of the sucklings, and 96 per cent. of the adults inoculated, were protected from the disorder. Inoculation is useless in cases of simultaneous sickness with swine-pneumonia and swine-fever, or severe tuberculosis. On the other hand, the polyvalent serum has succeeded in cases of co-existing mild swine-fever; when, besides the inoculation, a thorough disinfection of the sheds was carried out, as well as the slaughter of the animals affected with diarrhœa. The application of the serum has proved to be completely harmless. In the majority of cases a permanent protection was attained by a single injection. Inoculation has not proved effective in cases of sick animals, and should, accordingly, be omitted. In order to obtain the protection by inoculation in case of a non-infected suckling, inoculation should be carried out within the first three days of birth.

CHOLERA AND PLAGUE OF BIRDS.

Fowl typhoid—Fowl cholera—Fowl septicæmia.

History.—Next to diphtheritic inflammation of the mucous membrane, chicken cholera is the most important and most common disease of poultry. It is stated that it was known in ancient times, and was considered by some to be identical with the cholera or with the typhus of man; hence the name. Others regarded it as a form of anthrax. Lemaistre states that it was for the first time investigated with an attempt at accuracy in Lombardy during 1789, and that it raged in the East Indies in 1817. It has settled in France since 1825, and has caused during 1830 and 1850, and from 1860 to the present time, great losses in poultry. During the thirties it seems to have spread to Russia, Austria, and Bohemia, and during the last decades it has become common in nearly every European country. It has been for a long time the subject of veterinary investigation, and the knowledge of its symptoms, anatomical results, and mode of infection, was so far advanced, that only its bacteriology was left for modern research. As early as 1851, Benjamin assumed that a contagium was the cause of the disease and observed that men and dogs could consume the flesh with impunity. Delafond and others proved that the disease could be transmitted to poultry and rabbits by the inoculation of infected blood, secretions, and parts of the dead body; that retro-inoculation was possible; and that the fæces were connected

with the spread of the infection. Feeding experiments confirmed its contagiousness; and it was even then observed that the contagium lost its virulence when it became dry.

Modern investigations were inaugurated by a comprehensive work of Perroncito. It is very probable that Perroncito was the first to see the characteristic bacteria of fowl typhoid which Toussaint was the first to cultivate in neutral urine. Toussaint declared, later on, that these microbes were identical with the ordinary bacteria of septicæmia, against which supposition we have, besides other considerations, the fact that fowl typhoid cannot be produced by the inoculation of septic substances. Pasteur cultivated the fungus in sterilised chicken broth and first proved in 1880 that one attack conferred immunity. He also recommended protective inoculation. Thus, fowl typhoid was the first disease against which Pasteur used his method of protective inoculation, and was the starting point of his theory of protective inoculation. Kitt has been particularly prominent in recent bacteriological research. The schizomycete has recently been named bacillus avisepticus or *bacterium avicidum*, and has been relegated to the group of septicæmia hæmorrhagica (deer, and cattle-plague, swine-pneumonia, septicæmia of rabbits). On this account, the cholera of fowls has recently been also named fowls' septicæmia.

Bacteriology.—The contagium of fowl typhoid is an exceedingly small, ovoid, motile bacterium from 0·3 to 1 μ in length. These bacteria, which can be seen only when they are considerably magnified (immersion), are very short, biscuit-shaped rods of the form of the figure 8. They are constricted in the middle and translucent, or appear as oblong or circular globules. Under comparatively low powers, they can be seen only as minute double points. They stain well with aniline colours, but not with Gram's solution. The cultivations consist of small, hyaline, white dots, which appear in great numbers close to each other under the gelatine, and may attain the size of the head of a pin (colonies of fungi). Besides this, weak, dull-white layers with spherical dots form on the surface of the gelatine. The fungus grows best at a temperature of from 86° to 104° F. on neutral chicken broth, which is mixed with gelatine or agar-agar; and also on a solution of meat pepton, solution of extract of meat and sugar, hard-boiled white of egg, and solid serum. The bacteria can be transmitted to all kinds of birds, rabbits, and white mice, by cutaneous, subcutaneous, and per-

cutaneous [inunction] inoculation, and by feeding on blood, fæces, and parts of infected dead bodies. The inoculation kills in from 12 to 48 hours. In guinea-pigs, abscesses, which contain large numbers of the bacteria, form on the site of the inoculation (Pasteur). Only local abscesses form in inoculated sheep and horses (Kitt); and in man, when the contagium comes in contact with cutaneous wounds (Marchiafava and Celli). Kitt observed that an injection into the udder of a cow, set up catarrhal mastitis, and that the bacteria were found in the milk for a long time. Dogs and cats do not become infected by feeding on the bodies of poultry which have suffered from this disease; and apparently the consumption of the boiled flesh does not transmit it to mankind, although Zürn and Willach have officially reported two cases which, at least, suggest caution.

The bacterium of fowl typhoid is one of the easily destructible contagia. It is killed by mere drying, by most of the disinfectants, absolutely by boiling water, and by diluted sulphuric and hydrochloric acid (1 to 500). Kitt has found that it dies when exposed for three-quarters of an hour to a heat of from 113° to 122° F.; that it is not killed by a temperature of 6·8° F. lasting for 14 hours; and that it remains virulent for 3 months when associated with other bacteria. According to Massa, cultures continue to be very virulent for 2 months.

The bacillus avisepticus is an ectogenous saprophyte widely disseminated in nature; it is, for instance, found in the earth, in the intestinal canal of healthy pigeons; in the saliva and throat mucus of healthy animals and men, and in the water of pools. It displays several grades of virulence, and appears to attain a high degree by entogenous development in the bodies of animals.

Occurrence and Dissemination.—Fowl typhoid, as we have already said, attacks all kinds of birds, especially poultry, pigeons, peacocks, pheasants, parrots, and canaries. It frequently stops the breeding of poultry by destroying all the birds in a poultry establishment, and even entire breeds. The infection is communicated usually by the consumption of the fæces of infected birds along with the food or drinking water; and often by the feeding on the offal of infected birds which have been killed. It is generally introduced into a poultry yard by newly-purchased, infected birds; or by neighbouring birds, as for instance, pigeons which visit strange poultry yards. The importation of foreign poultry is a very common means of

spreading this disease. Thus, the cholera of fowl and that of geese were introduced into Prussia chiefly by Russian, Polish, and Silesian poultry. Bavaria and Southern Germany were infected principally by Italian poultry. The disease is often spread by poultry shows. According to Barthelemy, infection may take place even by feeding on the eggs of infected birds; because some of the bacteria which are within the body of infected hen-birds, may penetrate into the eggs, as has been proved to occur in the case of mammalian fœti (guinea-pigs). It is possible that the contagium may also be absorbed by wounds in the skin. Buchner has shown by experiment that the bacteria, on being inhaled, can pass through the intact surface of the lungs; thus giving rise to the disease. The infection readily occurs in animals exhausted by lengthy journeys (railway transport of geese from Russia). According to Theiler, fowl-cholera is sometimes stationary in South Africa.

Symptoms.—Fowl typhoid is characterised by the remarkable rapidity of its course, and by the extreme brevity of its period of incubation, which averages 24 hours, and has a maximum of 48 hours, and a minimum of 18 hours. In many cases death occurs apoplectically in such a sudden manner, that no signs of the disease are noticed during life. The birds are unexpectedly found dead in the morning, or they suddenly drop down dead off their perches. Hens have been known to die of this disease while sitting on their eggs.

In most cases, the disease lasts somewhat longer than we have just said; but on an average not more than from 1 to 3 days, and sometimes for only a few hours. The bird loses its appetite; is depressed and weak; droops its wings; blows out its crop; curves its neck; separates itself from its fellows; its plumage becomes ruffled; and there is a rise of internal temperature of from 2° to 6° F. We sometimes notice the discharge of frothy mucus from the beak, and vomiting. Diarrhœa, accompanied by violent thirst, sets in. The fæces are at first pappy and whitey-yellow, but later on they become mucilaginous, watery, green, and fœtid, and consequently, the parts near the opening of the cloaca become soiled. Dyspnœa soon sets in, and peculiar sobbing and wheezing sounds and rhonchi are frequently heard. The comb gradually turns blue. The bird becomes more and more debilitated, staggers, falls down, strives in vain to get up, and dies either comatose or convulsed.

In exceptional cases, and apparently when the infection has been present for a considerable time in a poultry yard, the disease may assume a comparatively slow course, and may then last a week or more. During feeding experiments, Semmer and others have seen death postponed for 3 weeks. Wertheim states that he has experimentally produced in pigeons, by the inoculation of somewhat old cultures, a chronic form of typhoid from which the birds took 10 days or longer to die, with symptoms of pyæmia; although fresh cultivations caused death from septicæmia in from 12 to 24 hours. According to Willach, the caseous masses which are found in the intestine, lungs, and liver, prove that in fowl-cholera a milder form also occurs, which runs a chronic course; and which may affect the bodies of the animals from 1 to 2 months, without their displaying any obvious symptoms of sickness. In this form the bacteria are much more sparsely distributed in the blood, and the infective power of the dung is much less.

Anatomical Conditions.—The principal changes are found in the intestines, heart, and lungs.

1. The external surface of the *intestinal canal* generally appears highly congested, and its mucous membrane, especially in the small intestine, is of a dark-red colour, and is covered with hæmorrhagic spots. The contents of the intestines may be watery, frothy, muco-purulent and yellow, or bloody and resembling chocolate. We also find defects in the epithelium, especially on the top of the intestinal villi, and sometimes even a croupy exudate on the mucous membrane, and the follicles of the intestines are often ulcerated. The mucous membrane of the colon, rectum, crop, and pharynx, is often greatly congested. The lymph glands of the peripheral group which is at the opening of the cæcum, is often swollen to the size of a bean, and takes the shape of the top of a mushroom.

2. The *heart*, as a rule, is covered with red points (subepicardial hæmorrhages), as if sprinkled over with dark-red dots, but sometimes the only change present is severe injection of the blood vessels. The pericardium usually contains a slight quantity of serous fluid, and myocarditic and pericarditic changes may also be observed.

3. The *lungs* are often found to be very rich in blood, thickened, of a dark brownish-red colour, and so heavy that they will sink in water (croupous and hæmorrhagic pneumonia). Fibrinous pleuritis and peritonitis have also been observed in a few cases.

Catarrhal laryngitis, tracheitis, bronchitis, and œdema of the lungs are not rare. The respiratory changes chiefly occur in land birds; and intestinal and cardial affections in water-fowl.

The other changes are in no way characteristic. We should remember that the flesh of birds which have died of typhoid is frequently normal in appearance, especially when the course of the disease has been very rapid. In other cases it is of a dark dun (grey-red) colour, and suffers from fatty and lardaceous degeneration. The liver sometimes shows dots. Dark-blue *post-mortem* spots often appear very quickly on the skin. The bacteria of typhoid are to be found in large numbers in the blood, and in all the organs of the body. In chronic cases, caseous deposits are sometimes found in the lungs and intestines as in contagious pneumonia, in the deposits of which, the ovoid bacteria can be demonstrated (Sticker).

Diagnosis.—The diagnosis of fowl typhoid during life depends on the epizoötic character of the disease; its exceedingly acute course; and frequency of diarrhœa. Ample data for the recognition of the disease after death are furnished by the morbid changes in the intestines, heart, and lungs, and by the presence of the bacteria. It is hardly possible to confuse the disease with diphtheritic inflammation of the mucous membrane, which has a much slower course. and which is characterised by the presence of a diphtheritic coating of the mucous membrane of the mouth. It is often more difficult to distinguish typhoid from acute poisoning, for which it is frequently mistaken by owners of birds. Such cases can be decided only by a *post-mortem* examination. According to Grimme, the *erysimum cripidifolium* (Reichenbach) produces, especially in geese, a form of disease similar to fowl-cholera (so-called geese-death). In the municipality of Merseburg, a number of fowl in one farmyard sickened with choleraic symptoms as the result of an infection with numerous entozoa (trichosoma collare).

Diagnostic Inoculation.—Kitt points out that the local anatomical changes which are observed in inoculated animals may be utilised for the diagnosis of fowl cholera. The constant characteristic change on the site of the inoculation in pigeons (which die in from 12 to 48 hours in consequence of the inoculation) consists in a yellow nodular prominence. After removing the skin, the surface of the muscles is found to be covered with patches of a dry, straw-coloured exudate, varying in size from a sixpenny piece to a shilling; and a transverse section of the muscles through the patches of exudate shows yellow discoloration and nodular induration. The site of the inoculation in

fowl manifests white, lardaceous discoloration, and swelling and induration of the muscles. The bacteria are found in large numbers by themselves in the blood contained in the heart of inoculated pigeons. The diagnosis can also be confirmed by feeding experimental animals with the diarrhœal discharges of the affected ones.

Prognosis.—The prognosis, contrary to what is the case in fowl diphtheritis, is very unfavourable in fowl cholera, in which the mortality averages from 90 to 95 per cent. In some cases, the disease runs a milder course ; probably because the contagium varies in virulence, and a certain amount of immunity is acquired by the bird having passed through a previous attack. Also, experience teaches us that early treatment considerably decreases the mortality.

Therapeutics.—The course of the disease is so rapid that we are often too late in administering medicines, which, apart from this, are of less importance than prophylaxis. It is always advisable to at once administer internally a ½ to 1 per cent. solution of sulphate of iron or of hydrochloric acid, or a weak solution of tannin (½ to 2 per cent.). We prescribe, for instance : ferri sulphas, 5 parts ; aquæ destillatæ, 500 parts ; or, acidi hydrochlorici, 3 parts ; aquæ destillatæ (fœniculi *vel* menthæ), 300 parts ; and we give a tablespoonful of either of these solutions every hour to geese and domestic fowl, and a teaspoonful to pigeons. These medicines may be administered prophylactically to healthy birds in an infected poultry yard. Nocard states that he has often cut short the disease by subcutaneous injections of a 5 per cent. solution of carbolic acid. Beck recommends creolin ; Buhl opium.

Prophylaxis consists in the separation of the healthy birds from the infected (not *vice versâ*), and in a thorough disinfection of the yard or coop, which may be carried out as follows : the floor, walls, utensils, perches, etc., are thoroughly cleansed either with a hot solution of caustic potash, or, still better, with a 1 to 1,000 solution of corrosive sublimate, or a 3 per cent. solution of carbolic acid or creolin. The walls are white-washed, and the dead bodies and all fæcal matters are burned or buried at a sufficient depth. It is advisable to repeat several times this method of disinfection.

Veterinary Police Regulations.—In Germany, the general duty of notification was ordered by the State on May 17, 1903. Special ministerial proclamations (1897 and 1898) laid down in Prussia the instructions for the management and driving about of the fowl and of the

Willerdung unfavourable ones. Jesz and Piorkowski have recommended a curative and a protective inoculation ; so also have Braun and Klett.

Cholera of Ducks.—Cornil and Toupet state that ducks suffer from peculiar infective diseases which are different from ordinary fowl cholera, although they resemble it in symptoms and anatomical changes; for death ensues in 2 or 3 days with diarrhœal muscular tremors, weakness, and paralysis. These diseases are characterised by the fact that the inoculation with the ovoid bacteria which are present in the blood, produces a fatal disease only in ducks, but not in other birds. Willach has in addition described a form of cholera, produced by ovoid bacteria in water-fowl (ducks, geese, swans), which is communicated, not by food, but by subcutaneous inoculation to poultry and pigeons.

Cholera of Canaries.—Besides the fowl-cholera which is produced by the *bacterium avicidum*, and occurs sometimes among canaries (see Vol. II., p. 261), other specific, cholera-like disorders are met with in these birds. Zürn was the first to call attention to it. Afterwards (1889) Rieck described a special disease of canaries, produced by ovoid bacteria, and accompanied in its course by multiple hepatic necrosis. Finally, Kern has observed a new infectious disease of canaries, which is produced by a special bacillus, and runs a fatal course, within 24 hours, accompanied by choleraic symptoms. The bacillus proves to be non-pathogenic to fowl and ducks; thereby contrasting with the bacterium avicidum and the bacterium of the cholera of ducks, respectively. Canaries, sparrows and other small birds, however, die after inoculation. On dissection there are inflammatory appearances found especially in the duodenum (swelling of the mucous membrane), a yellowish puffy thickening of the submucosa and punctiform hæmorrhages.

Klein's Fowl Disease.—Klein describes an avian epizoötic (grouse disease), which is independent of fowl cholera, and which is caused by a bacillus with rounded ends (*bacillus gallinarum*). It commences with diarrhœa and ends in death in 30 hours. Pigeons proved immune to the inoculations. Domestic fowl died in 3 days after a period of incubation of 6 days. On *post-mortem* examination the liver and spleen were found to be enlarged and softened. The very abundant intestinal mucus is almost a pure cultivation of the bacilli. Klein has recently described another bacillus, under the name of *bacterium phasianicida*, which belongs to the group of the bacilli of hæmorrhagic septicæmia, and which produces choleraic symptoms in pheasants.

Epizoötic Dysentery.—Lucet described under this name an infective disease that is said to chiefly attack domestic fowls and turkeys, and is manifested by depression, debility, complete loss of appetite, violent thirst, and profuse diarrhœa, which is at first mucilaginous, then bluish-grey, later on yellow, and finally sanguineous. The temperature falls 3° or 5° F. Most cases end in death, which takes place generally between the ninth and thirteenth day. This disease is supposed to be caused by a faculative bacillus, which can be easily cultivated on peptonised veal broth. It is distinguished from chicken cholera by its slower course,

its slighter infectiousness, and the impossibility of transmitting it by inoculation to rabbits. It has a period of incubation of 3 or 4 days. Lucet has also observed (1895) a choleraic disease of fowls produced by *bacillus enteritis gallinarum*.

Maladie du Sommeil.—Nocard gave this name to a disease of fowl which is caused by a very small bacillus. The bacilli are particularly numerous in the spleen. The disease was at first considered to be a chronic form of typhoid. It is characterised chiefly by stupor and almost uninterrupted somnolence. The infected animals die in from 8 to 14 days.

Vibrio-cholera of Fowl.—This disease, which has been described by Gamaleïa, is caused by a very motile, curved bacterium that has slender flagella (*vibrio Metschnikoff*). It closely resembles the cholera bacillus of man, on which account the name of "vibrio-cholera" or "gastro-enteritis cholerica avicum" has been given to it.

Mycosis of Parrots (Psittacosis).—Wolff described under this name an infective disease of parrots which has raged for the past 15 or 20 years among recently imported grey parrots (*psittacus erithacus*, and *jako*), and which kills them by thousands. The infection is said to take place on board ship during the voyage from the west coast of Africa. It is caused by micrococci, namely, by the *streptococcus perniciosus* (Zopf). Its development, according to Wolff, is favoured by damaged food, bad drinking water, dirty and unsanitary condition of the cages, and bad air in the holds of the ships. The symptoms of this disease, which generally ends in death, are: decreased appetite, debility, depression, drooping of the wings, diarrhœa, in rare cases vomiting, squatting on the floor, and finally convulsions. On *post-mortem* examination we find in the liver, and less frequently in the lungs, spleen, and kidneys, characteristic grey and dirty-white hard nodules which are about the size of millet seeds, and which contain the pathogenic micrococci in great numbers. Local necrosis is caused by the cocci settling in the tissues. Also, slight enteritic and sometimes lobular-pneumonic changes are met with.

Recently there has been described under the name *psittacosis* an epidemic form of pneumonia occurring in human beings, which is said to be developed as a result of infection by diseased parrots (?). This parrot disease, which is said to be communicable to man, manifests itself as an infective enteritis, which displays the presence of various micro-organisms (streptococci, staphylococci, pneumococci, bacteria, coli, etc.). A proof of the alleged association between parrot disease, and endemic pulmonary disease in man has no more been established than one of the earlier hypotheses of the origin of human diphtheria from the diphtheria of fowls.

Miscellaneous Observations.—Diseases of birds, similar in symptoms to those of fowl-cholera, have also been described by various observers. Lignière, San Felice and Martel found the *bacterium coli* in poultry and pigeons, and Joest the *bacterium intestinale gallinarum*, as the causes of an epidemic form of diarrhœa. Santori saw grave septicæmia

in fowls with the symptoms of fowl-cholera, and a special chromogenic bacterium. Moore and Dawson describe an infectious leucæmia in chickens (due apparently to the bacterium sanguinarium), which is often confused with fowl-cholera. Moore has also found a special bacillus in ailing doves which belongs to the group of swine-fever bacteria. Fiorentini determined the presence in pheasants of a specific septicæmia, produced by a special oval bacterium; also in swans a hæmorrhagic septicæmia. Publications of similar, in some instances rather questionable, discoveries in fowl have been made by Lisi, Perroncito, Mazza, Belfanti and Zenoni. Gabritschewski has observed a septicæmia spirochæte in geese (compare the chapter on Relapsing Fever—*Febris recurrens*).

FOWL-PLAGUE.

Historical Notes.—Under the names of bird-plague, fowl-plague, Brunswick bird-plague, new bird-plague, and Kyanolophia, a very destructive bird disease was described, in 1901, in various quarters at the same time; and which had till then been unknown in Germany. It had come from Italy, where it had been known for 10 years under the name of typhus (Centanni). In 1901, especially, it spread more widely in Northern Italy (Ferrara, Padua, Lombardy, Venice); passed into the Tyrol (Lode and Gruber); and was also imported into Germany, where in February, 1901, it first appeared at the Brunswick Fowl Exhibition (Jesz). Partly from Brunswick, and partly from Italy direct, it was then in that year distributed all over Germany. It was observed and described in Würtemberg (Lüpke), Oldenburg (Greve), Hessen (Joest); and as more widely spread in Prussia (Ostertag and Wolffhügel, Künnemann).

Etiology.—Fowl-plague is produced by some infective agent, which is not recognisable by the methods of research that we possess at present, and which is contained in the blood, dung, and nasal mucus. This infective material, invisible under the microscope, passes through the pores of a porcelain* filter, and has not yet been artificially cultivated. The disease is remarkably infectious. The infection and distribution take place through the dung and nasal mucus of the affected birds, and also through the blood and viscera of those which have necessarily been slaughtered.

The disease is easily transmitted from the affected to the healthy birds; by feeding; and by inoculation—subcutaneous, intramuscular, intravenous and intraperitoneal (blood, liver,

* Diatomaceous earth, which, after the destruction of organic matter by heat, is used as an absorbent powder,

spleen, kidneys, nasal mucus, intestinal contents). On the other hand, it cannot be conveyed to pigeons, ducks, swans, mice, guinea-pigs and rabbits. In exceptional cases young doves sicken after artificial infection, with symptoms of labyrinthine vertigo. The virus is destroyed in 40 hours by drying in the open air in sunlight. It is at once destroyed by heating to 158° F. It is also destroyed by the onset of decomposition and displays but slight resistance to disinfecting media. A 4 per cent. carbol solution kills it in 15 minutes; a 0.1 per cent. sublimate solution in 10 minutes; concentrated milk of lime, immediately.

Symptoms.—Fowl-plague has received its name from the fact that it specially, and often exclusively, attacks these birds. It causes death in 2–4 days, and thus can suddenly strike down large numbers of fowl. The chief symptoms are: dulness, ruffled plumage, a bluish-red comb, drowsiness, coma and general paralysis. Within the first 24 hours the birds display, as a rule, no change from the normal condition, or rise of temperature (incubation stage). On the second day, they appear less lively, and sit moping in a corner of the cage, as if brooding in a nest. The condition rapidly becomes worse in the course of the second day. The animals stumble in walking, and fall on one side, or forwards. The plumage is ruffled; the internal temperature rises to 111.2° F. Swallowing movements are frequently observed; also forced expiration and stertorous inspiration. The comb and gills are often of a bluish-red ("Kyanolophia"). The excrements are either of normal consistency and colour, or thinly fluid and green. The comatose stage usually sets in at the end of the second day; lasts for from 12 to 24 hours, and always ends in death. The birds can no longer move, and die with the appearances of general paralysis, with lowering of the body temperature. In many cases, however, spasms occur before the end; violent movements ("circus" and rotatory in form), unilateral paralysis and hiccough appear before death. In rare cases the fatal result occurs in from 6 to 9 days.

Anatomical Conditions.—No specific changes are found on dissection (Ostertag and Wolffhügel); especially no local ones in the intestine. Pathological changes are sometimes entirely absent. The cadaver usually displays merely the characteristics of a blood-poisoning; especially hæmorrhages in the mucous

membranes (alimentary canal, air-passages, oviducts), and the serous membranes (epicardium, abdominal cavity); and fatty degeneration of the liver (cloudy degeneration). Of local changes, the most frequent are hæmorrhages into the glands of the stomach. In many cases, also, serous fluid has been found in the abdominal cavity; in the pericardium; and œdema of the skin; superficial redness of the mucous membrane of the small intestine; but inflammation of the lungs and peritoneum have rarely been observed.

Differential Diagnosis.—Fowl-plague differs from fowl-cholera—in common with which it has the infective and endemic character, the rapidly fatal course, and the symptoms of weakness and paralysis—by the following important features: 1. By attacking fowls exclusively; 2. By non-communicability to old pigeons; 3. Negative bacteriological conditions; 4. Absence of diarrhœa and intestinal inflammation; 5. By a more prolonged course; while fowl-cholera often leads to sudden death; at all events, within from 1 to 3 days. Death never occurs so suddenly from fowl-plague, but takes place on an average after from 2 to 4 days.

Treatment.—Nothing is known up to the present of an effective medicinal treatment of fowl-plague, or of any method of inoculation. The spread of the disease can be prevented only by regulations enforced by the veterinary police. Such have been prepared in Prussia by the direction of the Minister of Agriculture. They coincide for the most part with those issued for the prevention of fowl-cholera (see Vol. II., p. 268).

DEER AND CATTLE DISEASE (*Septicæmia Hæmorrhagica pluriformis and Buffalo Disease*).

[Dr. Lingard (*Annual Report of the Imperial Bacteriologist* for 1902-1903) gives the following synonyms for this disease: *Barbone dei bufali* (Italy), *Wild und Rinderseuche* (Germany), *Pasteurellosis bovis* (France), Malignant sore throat (India), *Ghotwa*, *Gharrwa*, or *Galghotu* (Southern Punjab), *Khounnaq* (strangles) Egypt, *Sakit Ngorok* (Exanthemous) and *Koerang Naplas*, (pectoral form) Java. He tells us that of all animals experimented with, rabbits are the most susceptible to the organism of hæmorrhagic septicæmia.

"Animals which proved refractory to inoculated virulent cultures of hæmorrhagic septicæmia, viz., ponies, sheep, goats, and protected bovines and buffaloes, when subsequently injected for a long period with successively increasing doses of such cultures, furnished powerful protective sera against hæmorrhagic septicæmia" (*Lingard*).—TR.]

Occurrence.—Bollinger described under this name (*Wild und Rinderseuche*) an epizoötic which broke out during 1878 in the neighbourhood of Munich, and first occurred enzoötically among the deer of several royal parks, and later on also epizoötically, especially in cattle. The disease, which was at first confined to the deer and wild pigs that were kept in parks, soon spread to the cattle that were in the neighbourhood, and in some cases also to horses and domestic pigs. Altogether, 387 head of game died in the parks, namely, 153 red and fallow deer, and 234 wild boars. The epizoötic recurred in the following summers, and spread widely among the domestic animals of Upper Bavaria, especially during 1881. In subsequent years, it was restricted to certain localities. In 1889 it raged throughout the district of Bayreuth. It appears to have occurred among cattle in other countries, as for instance, in the Prussian district of Schlüchtern, where formerly there was an average yearly loss of 100 from it, but only of 11 in the year 1885-1886; also in Upper Silesia (1883). According to Jakobi, 34 out of 40 cattle which were grazing in the forests of Obornik died from it (principally from the exanthematic form) during the spring of 1889. In 1900, 14 cattle and one pig were affected in the districts of Memel and Schroda. Krüger-Schroda observed 118 cases in 18 parishes (101 cattle, 14 pigs, 3 horses). The disease is also known in Saxony (Eichhorn, Robert). Jochmann (Czarnikau) met with it in young cattle and pigs. During 1894, in the park of the Prince of Sigmaringen, 148 fallow deer and 25 other deer died of it (Hoffmann, Deigendesch). In Moravia the disease became known about 10 years ago; a wider distribution occurred there in 1900 (155 cattle, 29 boars).

This epizoötic was evidently well known in former times, as far as its complex symptoms were concerned. Thus, the "terrible" epizoötic among cattle and red deer which was described by "The Veterinarian" in 1854, was certainly this deer and cattle disease. It was usually mistaken for anthrax, with the lingual form of which it possesses in some cases great similarity. The question whether the majority of the cases described as "anthrax of the tongue" or "gloss-anthrax" were not those of this disease, is almost self-suggestive.

Etiology and Pathogenesis.—The deer- and cattle-plague is, according to Hueppe, Kitt and Nocard, a form of the so-called septicæmia hæmorrhagica or pluriformis, which is produced by the bacillus plurisepticus; and is identical with, or, at least, closely related to, swine-pneumonia, buffalo-plague, fowl-cholera, calf-septicæmia, etc. The bacillus presents a short, non-motile, girdled, bipolar, staining form (*bacterium bipolare*), of 0.6-1 μ in length, and 0.3 μ in breadth; it is found in enormous quantities in the blood and hæmorrhagic exudates. Gelatine cultures form whitish, hyaline and transparent droplets. Cutaneous or subcutaneous inoculation kills mice and rabbits after from 12 to 36 hours, with the appearances of bacteriæmia, hæmorrhagic laryngo-tracheitis (rabbits), and hyperæmic enlargement of the spleen (mice). Cattle, sheep, goats, pigs and horses also die very rapidly after inoculation. Friedberger and Hahn were the first to transmit by inoculation the disease from infected cattle to horses, which died from it, and also to cattle, which manifested only a local swelling. Friedberger and Frank found micrococci and small bacilli in the serum of the œdema. Bollinger successfully inoculated the disease in horses, goats, pigs, sheep, and rabbits. He produced in cattle the pectoral form by feeding with the sanious contents of the intestines of animals which had died in 54 hours from the exanthematic form, and produced the exanthematic form by inoculation of the blood and pleuritic exudate of animals which had died of the pectoral form; thus proving the identity of these two forms of the disease. No positive case of its transmission to man has been observed, and no bad results have been caused by wounds received during *post-mortem* examination, or by the consumption of the flesh of infected animals.

The exciting cause of the disease seems to be able to enter the body by the skin (exanthematic form), lungs (pectoral form), and intestines (intestinal form). Bollinger's inoculation experiments prove that the pectoral form may occur after the absorption of the contagium from the intestines. Wild pigs possibly become infected by eating the bodies of animals which have died from this disease. The exanthematic form appears to be caused chiefly by the penetration of the contagium into small wounds of the skin (stings of flies and hornets, injuries on the head and in the oral cavity). A direct transmission from one animal to another has never been known. The disease has also been disseminated by the sale of the meat and skins in

other villages. Zeiliger reported even a case of a man who had been employed to search for the carcasses of the deer that had died from this disease, introducing it into his own cow-shed.

The bacillus plurisepticus appears to be an ectogenous, ubiquitous saprophyte, which abounds especially in the earth, in dung, decomposing matters, saliva, throat mucus, and intestinal contents of healthy animals; and appears to become extremely virulent by entogenous development in the bodies of animals. Its power of resistance is but slight, as it is killed even by exposure to sunlight, or by drying.

Septicæmia Hæmorrhagica.—In 1885, Kitt found in the blood and portions of the bodies of cattle, horses, and pigs, which had succumbed in the neighbourhood of Simbach to an epidemic disease of obscure nature, bacteria of oval shape, non-motile, 0.6 μ in length by 0.3 μ in breadth; which stained only at the extremities, and resided specially in the blood-serum. Inoculation proved them to be pathogenic to mice, rabbits, doves, smaller birds, pigs, goats, horses, dogs, sheep and cattle; in the case of rabbits, a hæmorrhagic tracheitis usually took place. The bacteria displayed a similarity to those of rabbits' septicæmia, fowl-cholera, and swine-pneumonia. Kitt presumes that this disease is identical with deer- and cattle-plague; especially having regard to the similarity of the conditions of the pathological anatomy, and the communicability to the same animals. Besides, Kitt found in the examination of the blood of cases of deer- and cattle-plague carried out in 1878, bacteria which were very similar to those above described. Hueppe has confirmed those results in examination of the material forwarded to him by Kitt in connection with the above unknown disease, and obtained the same results on inoculation of the bacteria on smaller animals. He also holds these bacteria to be identical with those of deer- and cattle-plague; which, according to him, may manifest itself as a pure septicæmia, as an intestinal mycosis, and as an infective pleuro-pneumonia, and for which he proposes the name of "septicæmia hæmorrhagica." Hueppe has also inoculated rabbits, guinea-pigs and mice with cultures of the bacteria of swine-pneumonia, of the infective disease investigated by Kitt, of fowl-cholera, and of rabbits' septicæmia; and in all these cases he found absolutely identical changes, and identical action of the bacteria in the fluids of the tissues and in the blood. Accordingly he holds that swine-pneumonia, fowl-cholera, rabbits' septicæmia, deer- and cattle-plague, and also the contagious pneumonia of horses are identical diseases, and simply various manifestations of his "septicæmia hæmorrhagica" (?). According to Kitt we have, in case of the outbreak of any one or other of these diseases, to deal with distinct disease-forms and disease-agents; which, connected as they are by an arborescent relationship, appear, as the result of accommodation to environment, as varieties or races of ever-varying quality of virulence. Voges holds the same view, on the ground of his own researches. According to Nocard and Lignière, the bacillus suisepticus belongs to the Pasteurella genus (see Vol. II., p. 278).

A disease of calves similar to cattle-plague, and in which the same oval bacteria have been demonstrated, was described by Jensen. On one

cattle-farm in Jutland, 16 calves of about 2 months old, died suddenly, after a sickness of from 12 to 24 hours, with symptoms of diarrhœa accompanied by high fever (105.8° F.). Dissection displayed gastro-enteritis, fibrinous pleuritis and pericarditis, with numerous ecchymoses under the epicardium. Experimental inoculation of calves, rabbits and mice, produced an acute fatal septicæmia; the inoculated rabbits usually displayed hæmorrhagic tracheitis, and also great swelling of the spleen. Bongartz has also published a report on a similar disease of calves. Buch has observed 3 cases of sporadic hæmorrhagic septicæmia in adult cattle, accompanied by the same bacteriological conditions. Guillebeau and Hess have seen a form of hæmorrhagic septicæmia in cattle, which proved very destructive, with the aspect of an acute pneumo-pleuritis; and in which swellings on the head developed in some instances. The bacillus found was not, however, identical with that of cattle-plague. Bosso has found a form of septicæmia among cattle in Italy, with a predominant localisation in the kidneys. According to Leclainche, the cornstalk disease observed in America is identical with cattle-plague, but this has been controverted by Moore and Boström. Brimhall and Wilson observed in 1900 several enzoötic outbreaks of hæmorrhagic septicæmia among cattle in Minnesota, and on dissection recognised the bacillus plurisepticus in all cases. The most important symptoms were: swelling of the legs and of the lower jaw; discharge of blood with the fæces, urine, and nasal excretions. Death occurred in from 6 to 24 hours. Dissection displayed copious extravasation of blood in the subcutaneous tissue and in the internal organs. According to Blin and Carougeau, the disease which rages in Eastern Asia (Annam), under the name of "rinderpest," is no other than a hæmorrhagic septicæmia, of the group of Pasteurellosis, In large districts it swept away all the cattle and buffaloes, and ran both an acute course (with bloody diarrhœa, cough, salivation, lachrymation, hæmorrhages of every variety) and a chronic one (wasting, pulmonary lesions like those of epidemic pneumonia); while bacteriological examination displayed the presence of bacteria of the Pasteurella group. Quinine is said to be effective for its treatment.

Graffunder and Schreiber have observed a septicæmic and hæmorrhagic form of angina in pigs, produced by the bacillus plurisepticus. The pigs had great swelling around the windpipe and neck; salivation, lachrymation; dyspnœa; coughs; vomiting and fever (104° F.). Dissection showed a gelatinous, serous infiltration of the skin, subcutaneous tissue and muscular tissue of the head and neck as far as the thorax; lymphatic glands swollen to the size of a fist, and displaying necrotic foci of a size varying from that of a pea to that of a hazel-nut; gelatinous swelling of the mucous membrane of the throat; with diphtheritic foci, œdema of the lung, and sub-pleural hæmorrhages.

Pasteurellosis.—Under this name, Lignière has described an epidemic form of disease, which prevailed among Argentine cattle, horses and sheep, appearing partly in the form of a hæmorrhagic septicæmia (running an acute course), with the symptoms of pneumonia, pleuritis, peritonitis, etc., and partly also as a chronic disease, with diarrhœa, wasting and arthritis;

and caused by ovoid bacteria of the Pasteurella group. Lignière and Nocard have proposed the collective use of the term *Pasteurellosis* for all these infective disorders (instead of that of "hæmorrhagic septicæmia"), which are produced by bipolar cocci, non-motile, staining with re-agents; and which appear in very different varieties and forms of development. They have proposed the following classification of Pasteurellosis:

1. *Cattle:* Deer and cattle-plague; buffalo-plague; calves' diarrhœa; septic pneumonia of calves; Irish "white scour"; Argentine diarrhœa and "enquêté."
2. *Horses:* Influenza (horse-distemper) and epidemic pneumonia; especially all varieties of influenza.
3. *Sheep and goats:* Infective pneumo-enteritis and pneumonia; Argentine "lombriz."
4. *Dogs and cats:* Distemper; Stuttgart dog-disease.
5. *Pigs:* Swine-pneumonia.
6. *Fowl:* Cholera and diphtheria.

According to Kitt, the arbitrary re-baptism of hæmorrhagic septicæmia as Pasteurellosis has justified the protest against it, and has been repudiated even outside Germany.

Anatomical Conditions.—From the anatomical changes in the principal organs, we can distinguish three forms of "deer and cattle disease," namely, exanthematic, pectoral, and intestinal. The first two are the most important, and are frequently complicated with the third. In some cases, all three forms are present at the same time.

1. The *exanthematic* form manifests itself first by a considerable swelling of the skin and subcutaneous connective tissue. The skin is œdematously swollen on the affected parts (head, intermaxillary space, and neck), and the subcutaneous connective tissue is frequently sero-gelatinously infiltrated to an enormous extent, and thickened. In the meshes of the subcutaneous connective tissue, we sometimes find a clear, amber, or gold-coloured serum; at other times it is infiltrated with hæmorrhages of greater or less size. The lymph glands (submaxillary and upper cervical glands) in the neighbourhood of these swellings are partly serously, partly hæmorrhagically infiltrated, and are greatly enlarged. The mucous membrane and the submucosa of the oral cavity may show similar changes, in which case, they become cyanotic and considerably thickened. In particular, the mucous membrane on the sides of the tongue

often forms gelatinous, loose pads. The tongue itself is sometimes enlarged out of shape. On one occasion, Friedberger found the tongue of an ox to be 4 inches thick, and to weigh nearly 9 pounds. Its surface varied in colour from purple to a dirty brown-red, and under the epithelium were found dark red effusions which were the size of the palm of a man's hand, and which extended into the muscles of the tongue. The tissues of the tongue contained a large amount of juice, and a bright yellow fluid which resembled plasma and which rapidly coagulated, flowed from the purple-coloured section. The mucous membrane of the pharynx frequently shows similar changes. In these cases, the salivary glands appear to be very anæmic and dry. The respiratory mucous membrane may be swollen in the same way. Thus we find in the trachea, bronchi, and especially in the larynx, loose projections of the mucous membrane, which considerably narrow the lumen of these passages. Croupy changes have also been seen in the bronchi. Hæmorrhages are found in various organs, especially under the serous membranes, in the muscles, and in the lungs. The spleen is perfectly normal, except that its pulp is somewhat drier than usual. The blood is almost always of a normal colour and consistence. The perirenal connective tissue is frequently infiltrated with blood. Lastly, we almost always find symptoms of hæmorrhagic enteritis.

2. In the *pectoral* form, the interstitial tissue of the lungs is gelatinously infiltrated, and the lungs themselves are in a condition of hepatisation (croupy pneumonia). The pleuræ are inflamed, swollen and covered with sero-fibrinous and purely plastic deposits (*pleuritis serosa et sero-fibrinosa*). A fluid exudate of varying quantity (in one case 6½ gallons) is found in the thoracic cavity. Pericarditis and mediastinitis are also present. Also in this form, we find hæmorrhages everywhere in the body, and frequently hæmorrhagic enteritis. The spleen and the blood are normal.

3. In the *intestinal* form, the mucous membranes of the intestines, especially of the small intestine, and sometimes that of the stomach, are swollen; the epithelium is desquamated; the mucous membrane is infiltrated with hæmorrhages of various extent (enteritis and gastritis hæmorrhagica); and a thin and sanious fluid is contained in the intestines. These changes are usually complicated by appearances of the exanthematic or pectoral form. *Post-mortem,* we find only extremely slight changes in very acute cases.

Symptoms.—Two forms, the exanthematic and the pectoral, can be distinguished clinically.

1. The *exanthematic form* is the one which usually attacks cattle, and generally begins with loss of appetite and diminution in the secretion of milk. The internal temperature is very high, and varies on an average from 104° to 110° F. The soft parts of the head, especially of the face, intermaxillary space, dewlap, and neck, are swollen, often to an enormous size. The swelling is tense, hard as a board, hot, painful, and rarely yields to the pressure of the fingers. As a rule, it disfigures the above-mentioned parts in a very striking manner, and may attain a thickness of 8 inches. Symptoms of stomatitis and pharyngitis appear at the same time. Salivation is set up; a glassy mucous discharge flows in strings from the mouth; and the patient has difficulty in swallowing, and sometimes performs continued masticatory movements. Frequently the tongue hangs out of the mouth, is swollen to twice or thrice its ordinary size, is of a purple colour, and sometimes shows the impressions which the teeth have made on it. The mucous membrane on the sides of the tongue sometimes forms loose, gelatinous projections. Difficulty of breathing, and even actual suffocation, may occur in consequence of the great swelling of the oral and pharyngeal cavities. The other visible mucous membranes of the head are of a brown-red colour and infiltrated with hæmorrhages; and the vaginal mucous membrane may be greatly congested. Towards the end of the attack, excessive dyspnœa and colic set in. The patient strains to defecate, groans, frequently lies down, and may even pass croupy casts of the intestine along with the fæces, which, later on, take the form of severe diarrhœa. There is increasing weakness, the animal persistently lies down, and death takes place in from 12 to 36 hours; the minimum duration of the disease being 6 hours, and the maximum, 3 or 4 days.

2. The *pectoral form* is the usual one which occurs in wild deer. Exact observations of the disease are naturally wanting. The symptoms which were observed in some cases in cattle were, on the whole, those of a rapidly-running inflammation of the lungs with dyspnœa. Its duration is longer than that of the exanthematic form, and amounts on an average to 5 or 6 days, and may even extend to 8 days.

Prognosis.—The prognosis in cattle (no prognosis is possible with wild deer) is very unfavourable. According to Bollinger,

the mortality is decidedly higher than that of anthrax. Putscher saw only 9 recoveries among a total of 95 head of cattle; and Jakobi, only 6 among 40. Krüger, on the other hand, saw recovery take place in a great number of cases, with a prolonged course (of 118 cases only 57 succumbed). He has also, in contrast to other observers, more often seen the pectoral form occur in cattle (71 cases); in 2 cases, the duration of the disease in the exanthematous form, was of from 14 to 16 days, and he observed the development, as a sequela, of a tendovaginitis of the extensors at the carpal and tarsal joints.

Differential Diagnosis.—" Deer and cattle disease " has been confused chiefly with anthrax and pleuro-pneumonia contagiosa.

1. The similarity to *anthrax*, especially in pigs, consists in the carbuncular swellings of the skin and oral mucous membrane; development of hæmorrhagic enteritis; and occurrence of hæmorrhages in all the organs of the body. Sometimes anthrax also occurs epizoötically among deer kept in parks. Bollinger, for instance, calls attention to the epizoötic of anthrax which raged in 1874 in Grunewald, near Berlin, and caused the death of 2,000 head of deer. In spite of this similarity, " deer and cattle disease " can be easily and with certainty distinguished from anthrax by the following facts: absence of the bacillus of anthrax; absence of enlargement of the spleen and of the characteristic tarry condition of the blood, which are always present in anthrax; easy transmission to pigs, which are rather refractory to anthrax; difficulty of transmitting it to sheep, which can be inoculated with anthrax with the greatest ease; innocuousness of the flesh when consumed by man; and immunity of man to " deer and cattle disease."

2. Although " deer and cattle disease " is often confused with *pleuro-pneumonia contagiosa*, especially at the beginning of an outbreak, such a mistake may be avoided without fail by paying attention to the anatomical conditions. The lung changes in " deer and cattle disease " are of a perfectly uniform character, and are of the same age; thus differing from the irregular lung changes in pleuro-pneumonia, the course of which is much slower. According to Kitt, the period of incubation of " deer and cattle disease " is far shorter, and the rapidity of its dissemination is much greater. In pleuro-pneumonia we find only pectoral symptoms, which are frequently entirely absent in cases of " deer and cattle disease." Also, calves can

suffer from "deer and cattle disease," which differs from pleuro-pneumonia by the easy and typical manner it can be transmitted. Finally these diseases can be readily differentiated by bacteriological examinations (specific bacteria), and also by the effects of inoculation (cutaneous inoculation of rabbits). The confusion of the pectoral form of rinderpest with pleuro-pneumonia can produce dangerous results of a far-reaching kind, especially in those cases in which inoculations are resorted to, and which may cause very disastrous effects. According to Krüger, however, the croupous pneumonia of cattle is produced by the bacillus plurisepticus (?).

3. Mistakes may also arise with *malignant œdema*, which, however, can be transmitted only by subcutaneous inoculation; though "deer and cattle disease" can be conveyed by feeding and by cutaneous inoculation. The same remark applies to *quarter-ill*, which is sufficiently distinguished by its stationary and narrowly limited occurrence, and the crackling character of its tumours. There is no difficulty in distinguishing "deer and cattle disease" from *rinderpest*.

Therapeutics.—Up to the present, the treatment of cattle suffering from "deer and cattle disease" has been entirely unsuccessful. Friedberger tried in one case a subcutaneous injection of a 10 per cent. solution of carbolic acid, and the internal administration of salicylic acid, but with negative results. It might be well, as in quarter-ill, to make free incisions into the swellings, with subsequent application of strong disinfectants. The German veterinary police regulations for "deer and cattle disease" are the same as those for anthrax.

Buffalo Disease.—An epizoötic disease of buffaloes is known in Italy under the name of "barbone." It is chiefly found in the districts of Salerno, Rome, and Terra di Lavoro, and closely resembles "deer and cattle disease." It was first described from a veterinary standpoint by Metaxa in 1816, and was regarded, later on, as typhus, anthrax, or strangles. We are indebted to the more recent investigations of Oreste and Armanni for exact information respecting its pathology. Barbone occurs chiefly in summer among buffalo calves. It begins with high fever (105° to 107° F.), stupor, loss of appetite, characteristic swelling of the intermaxillary space, salivation, mucous discharge from the nose, swelling of the mucous membrane of the mouth and tongue, and excessive dyspnœa. The swellings, which may also appear on the abdomen, throat, face, and limbs, pit on pressure with the fingers, and do not crackle. If we make an incision into them, we find that they are produced by a yellow gelatinous exudation. In very acute cases, the

animals die in from 3 to 6 hours. Usually they fall down in from 10 to 24 hours, and die in convulsions. Recovery may take place, if the animal remains alive over 24 hours. The duration of the disease in a district is generally 9 or 10 days, and the mortality is 40 or 50 per cent. On *post-mortem* examination, the subcutaneous connective tissue of the swollen parts and the interstitial connective tissue of the muscles lying under them, are found to be infiltrated with a yellow gelatinous exudate. The spleen is unaffected. We find in the blood, ovoid bacteria, which very closely resembles the bacteria of swine fever, fowl cholera, and septicæmia of rabbits. The disease can be transmitted to horses, cattle, sheep, pigs, guinea-pigs, rabbits, pigeons, and turkeys, but not to dogs. The virus is found not only in the blood and exuded matter, but also in the saliva, urine, milk, and fœtal blood. Natural infection takes place by small injuries of the skin, alimentary canal, and less frequently by the lungs (compare its similarity to "deer and cattle disease"). The contagium is destroyed by drying, boiling water, 2 per cent. solution of carbolic acid, and 5 per cent. solution of sulphuric acid. Sanfelice, Loi and Malato have also established the fact of the existence of the buffalo disease in pigs and cattle in Sardinia. The infection is conveyed by the saliva and the nasal mucus. The bacteria which were found have also proved pathogenic to horses and sheep. Pease has observed the buffalo disease in Indian buffalo in the Southern Punjaub; cattle and pigs were also affected by the plague. According to Schein, buffalo disease was in Indo-China usually confused with rinderpest (so-called Indian rinderpest); from which it is, however, to be distinguished by the regular occurrences of inflammation of the lung and by the recognition of the bacteria of hæmorrhagic septicæmia. Grijns has observed the transmission of the buffalo disease from the dam to the fœtus. Oreste and Armanni recommend inoculation as a prophylactic measure. They succeeded in attenuating the virus by the inoculation of pigeons, and produced immunity in buffaloes by inoculating them, on three occasions, with very minute quantities of the blood of inoculated pigeons. They state that during the years 1888-91, inoculation was performed on 1,986 buffaloes, out of which only 6 per cent. died; although formerly from 45 to 50 per cent. succumbed to the disease taken in the ordinary manner. It seems from this that protective inoculation has excellent results.

Buffalo disease has been proved to have occurred in Hungary. During 1891, 4 per cent. of all the buffalo calves suffered from it in Siebenbürgen. In 1892 the disease attacked 237 animals, out of which 231 died (Hutyra). Reischig regarded this malady as identical with bovine "deer and cattle disease." Makoldy and Sequens, on the contrary, consider that it is swine fever; because, in the same farmyards, buffaloes and pigs simultaneously exhibited symptoms of swine fever, while cattle remained unaffected. Gál recommends injections of 5 per cent. solution of creolin, and the internal administration of creolin in doses up to 1¾ ozs. Under this treatment, 5 buffaloes out of 17 recovered. More recent researches on buffalo disease in Hungary were carried out by von Rátz, and have given the following results. It appears especially in the summer months and among the buffaloes at pasture, and attacks most frequently the young animals. Those over six years old are seldom affected. The infection takes place through the alimentary

canal, as well as through the skin; artificial infection is carried out most easily through an abrasion of the skin. Attempts at infection through the alimentary canal have been without result. The occasional occurrence of the disease with buffaloes stalled during the winter proves that the natural infection can also take place through the digestive apparatus—from the food and water. The disease runs its course with high fever (107.6° F.); great depression; swelling—at first œdematous, and afterwards solid—of the neighbourhood of the gullet, which travels upwards to the head and backwards till it covers the neck and shoulders; rattling breathing; cyanosis of the nasal mucous membrane; swelling of the tongue; tympanitis, etc. Death usually occurs within from 12 to 24 hours; sometimes even after from 6 to 7 hours. The mortality ratio is from 70 to 96 per cent. Dissection shows extreme infiltration of the subcutaneous and submucous connective tissues; general fibrinous peritonitis; swelling of the lymphatic glands; hæmorrhages on the pleura and in the larynx; and subepicardial and subendocardial extravasation. The bacteria found in the blood are almost identical with those of swine-pneumonia and of fowl-cholera. Of animals experimented on, the rabbit has proved most susceptible; guinea-pigs and pigeons display more resistance; while fowls and ducks are not affected. Subcutaneous inoculation killed horses in 20 hours; cattle in from 20 to 48 hours; pigs in from 20 to 24 hours, with the appearance of septicæmia and much infiltration at the seat of inoculation. Buffalo calves die after cutaneous and subcutaneous inoculation in from 24 to 36 hours. The symptoms of buffalo disease and contagious pneumonia, which sometimes develops simultaneously in pigs, are, according to von Rátz, not those of swine-pneumonia, but of buffalo disease. The buffalo disease stands nearest to the deer and cattle plague. Inflammation of the lung has not, however, been hitherto noticed in buffalo disease in Hungary, nor a communication of the disorder to cattle (this last has recently been alleged by Betegh and Matjas).

QUARTER-ILL.

Quarter-ill in cattle—Quarter-ill in horses.

History.—Quarter-ill (symptomatic anthrax, or black-leg) was described in former years, under various names, as a form of anthrax, and in France is called *charbon symptomatique*. It has long been known that this disease is not infectious, and that the meat is quite innocuous to man and beast. Even in former days, very exact clinical descriptions of the disease were made; as, for instance, that by Wallraff in 1856, which, as regards symptoms, is in no way inferior to our present knowledge of the phenomena of this disease. Also, Pfisterer, in 1870, came to

the conclusion that the nature of quarter-ill is different from that of anthrax. Feser and Bollinger have, however, been the first to teach with certainty, by their etiological researches, that quarter-ill and anthrax are two entirely different diseases. Feser fully investigated quarter-ill while studying anthrax in the Upper Bavarian Alps in 1875. He became acquainted with quarter-ill in 1860, and states that even at that time he found slender motile rods in the muscle juice. He points to small, thin, motile, serpentine rods which have rounded ends, as the cause of the disease. By the subcutaneous inoculation of the mud of certain places in the Alps in which the disease had been very common, he produced its characteristic symptoms in cattle, sheep, and rabbits. In the same year Bollinger discovered the bacillus of quarter-ill in the form of a thin, long rod with rotatory movements; and produced the disease experimentally in cattle, sheep, and goats by the subcutaneous inoculation of the blood of animals suffering from quarter-ill.

We are indebted to Arloing, Cornevin, and Thomas for the recent investigations on the nature of the virus of quarter-ill. They have fully described its bacillus, and have clearly demonstrated its biological relations. The introduction of protective inoculation against quarter-ill was the most important result of the inoculation experiments made by them. The very recent labours of Kitt, Kitasato, Ehlers, Ernst, Leclainche and Vallée, on the bacteriology of quarter-ill are worthy of mention.

Occurrence.—Quarter-ill is a stationary infective bovine disease, that is to say, it is confined to certain pastures, altitudes and sheds, where it remains throughout the year. It is specially active during the warmer months (June, July, and August), and occurs chiefly on swampy ground. Besides cattle, it also affects sheep, goats, but seldom horses and pigs. Well-known quarter-ill districts are: in Bavaria, the Upper Bavarian Alps and the Swabian district and Middle and Lower Franconia; in Prussia many localities in Schleswig-Holstein, *e.g.*, Apenrade, Hadersleben, Husum, Steinburg, North and South Dithmarschar districts, Tondern; in the Rhine Provinces, the districts of Düsseldorf (Kreis Mörs), Wiesbaden, Cologne, Treves, Aix-la-Chapelle. The disease often extends to Würtemberg, also into Baden (Mosbach, Mannheim); rarely to Saxony. In Switzerland, the Bernese Oberland especially; also the cantons of Grisons, Glarus, Freiburg, Unterwalden, Schwyz; in Austria, the Vorarlberg, Salzburg, the Tyrol, the Carpathian and Styrian

Alps; the districts of Scheibbs and Lilienfeld in Lower Austria and Ennstal in Upper Austria are the favoured homes of quarter-ill. Also in various districts of France (*e.g.*, Franche-Comté, Auvergne, Dauphiné, Limousin, Haute Marne); in Hungary (Siebenbürgen); in Belgium (West Flanders and Liége); Holland; Upper Italy; Russia; Norway; Algiers; South Africa, Australia, and America ("blackleg").

[Pease states that quarter-ill causes great loss during the rainy season in the swampy districts of the Punjab and North-West Provinces of India.—TR.]

As a rule, only cattle between the ages of 3 months and 4 years become affected. Calves under 3 months, and indigenous cattle over 4 years are rarely attacked. The reason of this, perhaps, is that the former are not exposed to the infection on account of their food being restricted to milk; and that the latter are mostly immune from having had the disease (Arloing). Moreover, calves are also much less susceptible to the artificial inoculation of the disease than full-grown animals. For instance, they can bear with impunity an amount of inoculation material which would certainly kill mature cattle. If foreign animals are put on the infected pastures, they may become infected at any age.

One attack confers absolute immunity. It seems that pigs, dogs, cats, rabbits, grey rats, mice (at least to a certain extent), and man are naturally immune to the disease. This statement agrees with the well-known fact that the flesh of cattle which had suffered from quarter-ill can be consumed by man, dogs, and pigs without any ill result. Animals which are immune to quarter-ill are also immune to malignant œdema. The inoculation of horses, asses, and white rats, produces only a local reaction in the form of a swelling. On the other hand, quarter-ill can be easily transmitted by inoculation to cattle, goats, sheep, and guinea-pigs. In some localities, for instance, in the Upper Bavarian Alps, quarter-ill occasionally occurs simultaneously with anthrax. Hence it was looked upon in former times as a precursor of anthrax.

Statistics.—In Germany 1,772 cattle and 3 horses became infected with quarter-ill during the years 1891 to 1893; in Baden, 375 cattle from 1886 to 1890; in Austria, 376 cattle in 1886; and in Switzerland, 533 in 1893. Schmitt reports that quarter-ill caused very severe losses in the district of Mörs (Rhenish Prussia), especially during the early eighties after the great inundations, so that during these years, 12 or 13

per cent. of all the young cattle died from this disease. In the canton of Berne, 1,728 cattle died from it during the years 1882 to 1884, thus showing a yearly loss of £5,000 during that time. In the Transvaal, quarter-ill, which is locally known as *spons ziekte* (sponge disease), *okapiranka*, or *ongamero*, is said to occur throughout the whole year, and to kill 50 per cent. of all the young cattle (Theiler and Sander).

Bacteriology.—The bacilli of quarter-ill (*bacilli sarcophysematos bovis*) are very small, being only from 3 to 6 μ in length and 0.5 to 0.7 μ in thickness, and are therefore about the length of the diameter of a red blood-corpuscle. They show lively, rotatory, or serpentine movements, and contain, usually at one end, a bright oval spore, the greater thickness of which makes the rod assume the shape of a club. Contrary to the behaviour of other bacilli, they can be stained with Gram's solution. They are found most abundantly in the affected subcutis and muscles, but seldom in the blood; hence inoculations with blood are generally negative. They occur in great quantities in the bile and in the contents of the intestines (Kitt). In the blood, they are only found when the dead body has been kept for a considerable time. Consequently, the bacillus of quarter-ill, like that of malignant œdema, is an anærobe, which forms gas while growing in the tissues; it does not require for its development the oxygen which is in the blood; and it forms spores within the dead body (endogenous development). It is probable that it also develops ectogenously in the soil, although this supposition has not yet been positively proved. Arloing was the first to obtain a pure culture of the bacillus, which he did in chicken broth. Pure cultures are characterised by the abundant formation of gas; thus producing a large quantity of pearly bubbles and froth, which clear away in from 1 to 3 days, and leave a thick white sediment. The agar cultures are also very typical. By breeding in an incubator after from 12 to 36 hours, punctiform, glandular, delicate colonies are developed, with so copious a formation of gas that the nutrient soil is severed, broken up, raised and elevated, and the plug of wadding of the incubator is forced out; the gas which develops is odourless. On potatoes the quarter-ill bacilli, as is usual with the anærobic forms, are not developed. The bacillus flourishes best at from 96.8° to 100.4° F., but still grows at from 56.8° to 64.4° F. The higher the temperature, the faster the spores form. In the flesh and bile of the cadaver, a copious spore-formation takes place even a few hours after the death of the animals.

The investigations of Arloing, Cornevin, and Thomas have

shown that the virus of quarter-ill is exceedingly resistant to external influences, especially when it has been dried, in which condition it will continue effective for a couple of years. Hence the dried flesh remains infectious for a very long time. Burying the dead body does not destroy the contagium even in 6 months. The bacilli of quarter-ill is in no way influenced by the bacteria of putrefaction, by those of anthrax, or by severe cold. The dried and finely-powdered flesh of animals which have suffered from quarter-ill can resist steam even at 212° F., which degree of heat only weakens the virus, but does not destroy it. On the other hand, the fresh virus is destroyed by a temperature of 212° F. in 20 minutes, and by boiling water in 2 minutes; but the dried virus is destroyed only by a temperature of 230° F. in 6 hours, and by boiling water in 2 hours. The dry virus is also destroyed by the vapour of bromine; watery solution of corrosive sublimate (1 to 5,000), salicylic acid (1 to 1,000), thymol (1 to 800), carbolic acid (1 to 50), boric acid (1 to 5), hydrochloric acid (1 to 2), etc. On the other hand, these have proved ineffective: alcoholic carbolic acid solution, quick-lime, ferrous sulphate, zinc chloride, sulphurous acid, and other media, which have been regarded as potent disinfectants. Thus the fact has been established that the bacilli and spores of quarter-ill are weakened in resisting power by culture.

The more recent researches of Leclainche and Vallée have finally established that the toxin of quarter-ill bacilli displays a poison so deadly that, on intravenous inoculation, it kills a horse in 5 minutes; and guinea-pigs die in a few hours from intra-peritoneal or intra-muscular inoculation. Spores which have been made free of toxin are harmless; the spores being no longer protected by the toxin from the exterminating phagocytes.

Pathogenesis.—Quarter-ill is a wound-infection disease, that is to say, an infective disease due to the absorption of the bacilli of quarter-ill into the body by injuries of the skin or mucous membrane. These injuries, in order to afford the bacilli entrance into the body, must, as in the case of the bacilli of malignant œdema, penetrate into the subcutaneous connective tissue or submucosa; for mere penetration of the skin or mucous membrane is insufficient. It is supposed that such injuries are inflicted, especially on the legs and mouth, while the animal is grazing, and that they come in contact with the contagium, which is present in the soil. Hess found injuries of the skin on

the pasterns and cannon bones in animals suffering from quarter-ill, and is of opinion that infection of the injured oral mucous membrane takes place when grazing, chiefly while the milk-teeth are being shed. Hafner states that the oral and pharyngeal cavities form a frequent gate of entrance for the bacilli, particularly in stall-fed animals. According to the inoculation experiments of Arloing, Cornevin, and Thomas, an infection from the cow to the fœtus can take place. The calves of cows which have had quarter-ill seem to be immune.

The German veterinary reports attribute the frequent outbreaks of the disease to the inadequate disposal of the bodies of animals which have died of quarter-ill.

Inoculation experiments have shown that the period of incubation averages 2 days, with a minimum of 1 day, and a maximum of from 3 to 5 days.

Symptoms.—Quarter-ill has a very rapid course, which, with few exceptions, ends fatally in from one and a half to three days. It is characterised by a rapidly increasing swelling of the skin, which crackles on being touched; high temperature; secondary swelling of the lymph glands of the skin; and secondary motor-disturbances.

1. *The swellings of quarter-ill* may appear on different parts of the body, and are chiefly found on the upper parts of the thighs, neck, shoulders, lower region of the chest, loins, and sacrum. They never appear below the hocks and knees. They are less frequently found on the palate, at the base of the tongue, and in the pharyngeal cavity. The swelling is at first very small and painful. It rapidly spreads, may attain extraordinary size in a few hours, and may even extend over the whole trunk. These swellings are then characterised by a crackling and gurgling sound when the hand is passed over them or when they are kneaded with the fingers. They give on percussion a clear, tympanitic sound, and in their centre are insensitive, very dark-coloured, dried up like parchment, or even gangrenous. They become strikingly cold, and no pain is felt by the animal when an incision is made into them, in which case a dark-red frothy, unpleasantly smelling fluid flows from the wound. Sometimes, only one swelling of greater or less size appears; but more frequently we find several, which not uncommonly become confluent. The lymph glands in their neighbourhood are greatly swollen and can then be distinctly felt as tumours under the skin.

2. The chief *general symptoms* are as follows: sudden sup-

pression of appetite and rumination, debility, depression, high internal temperature (up to 107° F.), lameness, stiffness, and dragging of one leg in consequence of the swelling spreading to a limb. According as the swelling becomes larger, the other symptoms increase in intensity. The breathing becomes accelerated and difficult, and the animal groans, and is sometimes seized with violent attacks of colic. Death occurs almost invariably; and, as a rule, after the disease has lasted from 1½ to 3 days, with increased weakness and fall of the internal and external temperature of the body.

In some cases the general symptoms are the first to appear; in others, the characteristic swelling of quarter-ill takes the initiative. A few mild cases, which showed slight swelling and moderate fever, and recovered in 24 hours, have been observed in aged animals. Arloing mentions a mild form of quarter-ill which is chiefly manifested by loss of appetite, slight colic, tympany and debility.

Quarter-ill in the Horse.—Ganter has observed a horse die—in a locality in which quarter-ill is always present among cattle—in the course of 24 hours, with symptoms of high fever, perspiration, great psychic depression with vomiting and dyspnœa. On dissection there was found a brawny, bloody, gas-holding infiltration of the neck and left side of the breast, with hæmorrhage in the muscles and enlargement of the spleen. The fluid of the brawny infiltration of the neck contained the characteristic quarter-ill bacilli, the presence of which was established by Hafner. Other cases of quarter-ill in horses have been observed by Liebold, Leonhardt, and others.

Quarter-ill in Pigs.—Cases of this disease occurring in pigs have been described by Marck and Battistini, and appeared with the symptoms of pharynx-angina and of paralytic gait. Dissection displayed great swelling of the neck and the neighbourhood of the larynx and legs; brawny and serous infiltration of the transcular peripharyngeal and other tissues, dark-brown colouration and crepitation of the muscular tissue; with quarter-ill bacilli—with and without spores—in the muscles. Three rabbits inoculated by Marck died of inoculation quarter-ill (!). On the other hand, a pig and a sheep inoculated with the diseased flesh did not develop quarter-ill. Marck thinks that the infection in the pigs which suffered from symptoms of pharynx-angina took place through either the superficially injured mucous membrane of the pharynx or the uninjured tonsils. He also considers that quarter-ill is of not infrequent occurrence in pigs, although an epidemic prevalence has not been observed.

Anatomical Conditions.—The *skin* covering the swellings of quarter-ill shows the already described changes of dry gangrene. The subcutaneous connective tissue is yellow, gelatinous, infil-

trated with blood, and contains bubbles of gas, which escape if an incision be made into the tumour. The muscles underneath the tumours are of a dirty brown or even of a black colour. At other places they are dark red or dark yellow, and when exposed for some time to the air, they may have a golden lustre. They are brittle, rotten, porous, and very rich in fluids. They crackle on palpation or when incisions are made into them, and blood of a frothy, greasy, tarry condition and of a sickly fœtid smell issues from them when they are squeezed. The fibres of the muscles show extremely varied degenerative changes. The gases that are present in the muscles are inflammable and burn with a bluish flame on being ignited. They are stated to have but little smell, or to possess an odour which is not particularly bad; on which account it is assumed that they consist of carburetted hydrogen. They are also said to contain carbon dioxide, but no oxygen. An exact chemical analysis of these gases has not yet been made, although it would be very desirable. As some observers state that stinking gases are given off when incisions are made into the muscles, it is not improbable that the muscles also contain sulphuretted hydrogen. The lymph glands near the tumour are enlarged, and full of blood, contain hæmorrhages, and are sero-saniously infiltrated. The afferent lymph vessels are sometimes distended with gases, and then have the appearance of strings of beads. We find changes, similar to those of the external muscles, in the muscles of the tongue and pharynx, when the disease, as may happen in rare cases, is localised in the oral and pharyngeal mucous membranes.

In the *abdominal cavity* we frequently find a large amount of blood-red exudate; but in other cases, only small quantities of a serous fluid, or even no changes at all, according as the swelling of the muscles has or has not spread to the peritoneum. Yellow gelatinous and hæmorrhagic infiltrations are often met with in the omentum, mesentery, and neighbourhood of the kidneys. The mucous membrane of the stomach and small intestine is frequently swollen, congested, and infiltrated with hæmorrhages, in which case the contents of the intestine are bloody. The liver is hyperæmic, but the spleen is perfectly normal.

In the *thoracic cavity* we sometimes find the pleuræ in the neighbourhood of the swollen parts of the skin and mediastinum to be hæmorrhagically infiltrated. The pleuræ also shows large ecchymoses, in which case the thoracic cavity contains a sero-sanious exudate. Hæmorrhages are also present in the lungs,

pericardium, myocardium, and under the endocardium. The muscular tissue of the heart is very soft, but the other muscles show only slight changes. The mucous membrane of the bronchi are sometimes hyperæmic and hæmorrhagically infiltrated.

The *blood* is of a normal colour and readily coagulates. The fluids of the muscles have, according to Feser, an acid reaction, and the flesh becomes rapidly putrid. The dead body is greatly inflated by the gases that accumulate under the skin. The bacilli of quarter-ill are found only in small numbers in the blood during life, but abundantly a few hours after death.

Differential Diagnosis.—We have here to consider only the differentiation of quarter-ill from *anthrax*. With respect to the differential diagnosis of malignant œdema, see Vol. II., p. 153.

The chief diagnostic characteristics are as follows :—

1. Quarter-ill is *clinically* distinguished from anthrax by its characteristic crackling and gas-containing swellings, which never occur in anthrax ; by its non-contagious nature ; and by the greater severity of its course.

2. It is *anatomically* distinguished by the above-described changes in the muscles, which always contain gas, and by the normal condition of the blood of the spleen. It is also stated that the blood of quarter-ill, contrary to what is the case with the blood of anthrax, always coagulates completely.

3. It is *bacteriologically* distinguished by the bacilli being different. Those of quarter-ill are shorter and thicker, have rounded ends, are very motile, contain spores at one end, and are consequently club-shaped. The bacilli of anthrax, on the other hand, have square ends, are of uniform thickness, are non-motile, and have characteristic pure cultivations.

4. Cutaneous *inoculations* with the virus of quarter-ill give negative results ; but even a very small quantity of the virus of anthrax introduced into a wound of the skin is effective. Subcutaneous inoculations of quarter-ill cause on the site of the inoculation a very severe swelling, which never occurs to the same extent with inoculations of anthrax. Intravenous inoculations of quarter-ill produce only a slight affection with subsequent immunity, but similar inoculations of anthrax cause certain death. Guinea-pigs always succumb to inoculations of both anthrax and quarter-ill. Rabbits are immune to quarter-ill, but are exceedingly susceptible to anthrax. In order to be able to positively distinguish anthrax from quarter-

ill, we should inoculate simultaneously a rabbit and a guinea-pig. If only the guinea-pig dies, the disease is quarter-ill; if both die, it is anthrax. The predisposition to quarter-ill in animals is generally less than that to anthrax.

False Quarter-ill.—Various infective diseases have in recent times been confounded with quarter-ill—partly on account of the similarity of the clinical aspect, and partly from that of the bacteriological conditions. Foremost among these we have the puerperal quarter-ill, which is really a puerperal malignant œdema, or septic gas-phlegmon of the vulva and vagina; *i.e.*, a septic puerperal metritis, and which but exceptionally displays a pure quarter-ill (compare Septicæmia Puerperalis). Bacteria similar to those of quarter-ill have also been found by Kerry, Piana, Galli-Valerio, Klein (false quarter-ill), Nielsen (whale's quarter-ill—from infected harpoons), Nielsen and Jensen (Bradsot-epidemis gastromycosis of sheep), Lundgren and Bergmann (reindeer plague), and others. A definite distinction between these cases and pure quarter-ill is often difficult even for the expert bacteriologist. Of the greatest importance in differential diagnosis is the fact that pure quarter-ill occurs among cattle only, as a general rule, in the so-called quarter-ill districts; while animals above 4 years old are usually not afterwards affected.

Therapeutics.—Quarter-ill is such a malignant disease, and its course is so very acute, that treatment is generally out of the question; although, if we take the case in hand at the beginning of the attack, we may attempt a cure by making free incisions into the swollen parts of the skin, and by introducing strong disinfectants into the subcutaneous connective tissue. Wallraff states that he has heard of animals being saved, when the disease starts in a leg, by applying, as soon as the swelling begins, a tight ligature around the limb just above the enlargement, which should then be scarified. We cannot deny the possibility of a favourable result from this treatment. Tisserand tells us that, during 1877 in France, 32 head of cattle recovered from quarter-ill out of a total of 1,875 (not quite 2 per cent.). Arloing has lately recommended the serum-therapy.

The chief *prophylactic means*, if practicable, is to avoid those pastures which have been known to have produced the disease. In Upper Bavaria, for instance, a pasture land which was thus infected was converted into a forest (Zeilinger). As infection usually takes place by wounds of the skin and not from the intestine, Kitt proposes to feed the cattle in sheds with the hay made from the grass of infected places, and to exclude from pasture lands, cattle which have wounds on their pasterns or which are shedding their milk-teeth. It is, however, true that the disease not uncommonly occurs in cattle which are exclu-

sively stall-fed, as in Baden and Würtemberg. The best means of combating quarter-ill is the application of effective veterinary-police regulations. It may be reasonable to apply to quarter-ill almost the same regulations as those for anthrax, as has been done in several countries, for instance, in Prussia, Würtemberg, and elsewhere. In Austria, the following special measures have been put into force: compulsory notification; prohibition of slaughter; prohibition of using the dead body, with the exception of the skin; rendering the dead body and the offal innocuous; disinfection; and periodical inspection.

Protective Inoculation of Quarter-ill according to Arloing, Cornevin and Thomas (*Lyons method*).—The protective inoculation method introduced by Arloing, Cornevin and Thomas is of great scientific interest and practical importance. These authorities found that, although the subcutaneous and intramuscular inoculation of the virus almost always causes death, intravenous and also tracheal inoculation produces only a harmless general affection which is followed by immunity. On the other hand, all feeding experiments which were made gave no result. Working by the light of these observations, they inoculated 13 animals at Chaumont in 1880, by injecting, directly into the jugular vein, filtrated quarter-ill virus which had been dissolved in distilled water, with the result that the animals thus protected proved, 6 months later, to be quite immune to a subcutaneous inoculation of the virus; whilst out of 12 non-protected animals which were also subcutaneously inoculated with the virus, 9 died of quarter-ill and 2 became extremely ill. In 1881 they inoculated, with a similar good result, 245 animals in the Département de la Haute-Marne; and 78 during 1882, in the Département de l'Ain.

The intravenous method was found to demand such great attention to detail—for the jugular vein had to be laid bare on each occasion, and very exact precautions had to be taken when introducing and withdrawing the syringe, to avoid the penetration of the inoculation material into the perivascular connective tissue—that they discarded it for inoculation of the subcutis of the tip of the tail; because they found that this method produced only a temporary, harmless swelling, which was also followed by perfect immunity. They explain that the mild character of the reaction at the tip of the tail is due to the density of the connective tissue at that part, and to its low temperature, both of which facts more or less check the dissemination

of the bacilli in the connective tissue spaces. Arloing, Cornevin and Thomas also attenuate the virus by the influence of heat before inoculating. The inoculation experiments made during the last few years in France, Switzerland, Austria, Germany and other countries indisputably prove that Cornevin's protective inoculation greatly reduces the mortality of quarter-ill.

Preparation of the Inoculation Material and Inoculation according to the Lyons Method *(Double inoculation at the tail).*—The Lyons method is as follows: 1¾ oz. of infected muscle are rapidly dried at a temperature of 90° F., and are intimately mixed with 2⅜ oz. of water. The mixture is divided into 12 equal parts, which are placed on separate flat plates. These plates are put into a thermostat for 6 hours to dry, 6 of the plates being exposed to a temperature of 212° F. in order to produce the weaker or first material, and the remaining 6 to one of 185° F. to obtain the stronger or second material. The inoculation is made in two stages, first with the weaker, and afterwards with the stronger material. The dried-up brown crust on the plates is used for the inoculation, and may be kept for a moderately long period. For the first inoculation, 1½ grs. of the crust which was dried at a temperature of 212° F., is rubbed down in a sterilized dish with 85 mins. of water, so as to form the contents of a 5 grm. (85 mins.) Pravaz syringe. This syringe is manufactured according to Cornevin's designs by Lépine, of Lyons. The mixture is filtered through damped clean linen, and the filtrate is drawn into the syringe. The quantity of material required for each animal is 8½ mins. (0.5 c.c.); hence a syringeful suffices for 10 animals. The site of the first *inoculation is on the lower surface of the tail, at about a foot from the tip.* After the hair has been cut off, the skin is obliquely pierced with the trocar, which is supplied with the syringe, and the trocar is then pushed upwards for about 3 in. between the skin and the bone. After its removal, the cannula of the syringe is introduced into the same wound, and the inoculation fluid, which is previously shaken up in the syringe, and thus uniformly distributed, is slowly injected. To prevent the escape of the fluid, the site of the puncture is compressed after the cannula has been removed, and the injected fluid is pressed farther up under the skin with the thumb. If there has been any hæmorrhage, we must wait until it stops before we inject, or we must choose another site. On the site of the inoculation we finally place an indiarubber band about ¾ in. broad, and retain it in position for four hours in order to prevent the flowing out of the injected material. It is stated that from 20 to 25 animals can be inoculated in this way in an hour. The animal is held by three assistants. Ten days after the first inoculation, the second one is made with the stronger material, and is carried out in the same way, except that its site is at about 8 inches from the tip of the tail. The spring or the end of winter is the most favourable time for the inoculation, which produces no secondary disease.

Statistics of the Inoculation of Quarter-ill according to Arloing.—1. In 1883 Cornevin inoculated with great success in *France*, according to the Lyons method, 125 animals, and also obtained

very favourable results in 1884. According to Tisserand, inoculations were made in 1887 on 3,219 cattle, of which only 12 head (0·37 per cent.) died; and in 1888 on 2,086, of which only 3 (0·14 per cent.) died during the year. On the other hand, out of 4,000 non-inoculated animals, 50 (1·78 per cent.) died.

2. In *Switzerland.*—Following the example of Cornevin, Hess and Strebel made a large number of protective inoculations. Strebel states that over 2,000 cattle were inoculated up to the end of 1884, and were then kept during the whole summer on hills which were greatly infected by quarter-ill, with the result that the mortality of the inoculated animals was 28 times less than that of non-inoculated ones. He reports that 8,641 animals were inoculated in Switzerland during the years 1884–1888 with a loss of 15 (0·17 per cent.), and that 491 died of quarter-ill out of 21,000 (2·34 per cent.) which were not inoculated, and which had been kept under the same conditions as the other animals. Hence the mortality among the latter was 14 times more than among the former. He also says that the loss in 1889 among the non-inoculated animals in the canton of Freiburg was 12½ times greater than that of the protected 6,616, and 8 times more in 1890. The proportionate average loss among the inoculated and non-inoculated animals for the seven years from 1884 to 1890 is consequently 1 to 11½. He states that during 1885, in the canton of Bern alone, 15,137 cattle were inoculated with very satisfactory results; for deaths from quarter-ill decreased very considerably in the infected districts. For instance, in 2 villages in which this disease raged, the mortality of the protected animals was 8 times, and in another village 5 times less than that of the non-inoculated. According to Hess, 148,569 cattle were inoculated during the ten years from 1885 to 1894 in Bern with a loss of only 5 *pro mille*. With respect to the method of inoculation, Strebel recommends injection in the region of the shoulder, especially in front of the shoulder-joint.

3. In *Austria* Sperk, during 1885, inoculated 925 animals with extremely good results; for although these cattle were afterwards sent to graze on notoriously infected summer pastures of the Tyrolean Alps, not one of them, so it is stated, died of quarter-ill. Three calves, however, died after the first inoculation. On the other hand, out of 6,387 non-inoculated animals, 107 died of the disease. During 1886, 2,140 young cattle were inoculated in Salzburg, and 3,820 in Tirol-Vorarlberg, with a loss of, respectively, 4 and 16; while at the same time, out of 9,160 non-inoculated animals in Salzburg, and 17,401 in Tirol-Vorarlberg, respectively, 86 and 330 died of quarter-ill. The mortality of the non-inoculated cattle, is consequently from 1 to 2 per cent., and that of the protected from 2 to 4 *pro mille*. The cost for inoculating each animal amounts on an average to from *6d.* to *8½d.* In Salzburg, 2,472 protected, and 3,561 non-inoculated cattle were depastured during 1887 on hills infected with quarter-ill, with the result that 6·3 per cent. of the non-inoculated cattle, and only 0·3 per cent. of the protected ones, died. According to this, the non-inoculated animals are twenty times more liable to become infected than the protected. In 1888, according to Suchanka, 0·16 per cent. of 1,773 protected cattle, and 1·74 per cent. of 3,036 non-inoculated cattle became affected. The losses during 1889 in the same place were 0·16 per cent. among 1,222 protected cattle, and 1·77 per cent. among

5,616 non-inoculated. According to Wildner, there was only one death from quarter-ill in Lower Austria among 1,011 protected animals (=0·01 per cent). On the other hand, in 1896, 53 of 6,000 inoculated cattle died (=0·9 per cent.); so that people were officially warned against the use of the Lyons vaccine. In *Hungary*, during the years 1891 to 1893, only 5 died out of 3,361 protected cattle. In 1895, of 2,276 inoculated cattle, only 3 died; in 1896, of 1,100, none; in 1897, out of 2,469, but 4; in 1899, the total loss among 2,792 inoculated cattle was but ½ per cent.; and in 1900 3,434 cattle were inoculated with remarkably favourable results.

4. In *Prussia*, in 1886, 64 cattle were inoculated without any loss; in 1887, 485 cases were inoculated, while 264 non-inoculated were left as subjects of control-experiment. Of the inoculated, 2 died of quarter-ill; of the non-inoculated, 3—that is to say, almost three times as large a proportion as those inoculated. In 1888, 646 animals were inoculated; while 437 were left uninoculated as "controls." From the spring to the autumn of 1888, none of the inoculated animals were infected; while 4 of the non-inoculated died of quarter-ill. According to a decision of the Technical Committee for Veterinary Matters in Prussia (1901), the inoculation process is advised, as it produces immunity, and involves little or no danger. It is not, however, to be recommended in those localities in which quarter-ill seldom appears (*e.g.*, West Prussia); as the infection matter may be distributed by that of the inoculation; especially that of the second vaccine.

5. In *Baden* during 1886 to 1894, only 3 died out of 3,567 protected animals. The consequence of these extraordinary good results is that in Baden an indemnity is paid for fatal cases of quarter-ill, if it can be proved that the animals had been inoculated.

6. In *Italy*, in 1895, there were 20,000 cattle and 13,000 sheep inoculated against quarter-ill; in 1896, 20,000 cattle and 34,000 sheep; in 1898 and 1899 (together), 120,000 cattle and 260,000 sheep. In *Sardinia*, the centre of quarter-ill, all animals are inoculated.

Kitt's single Inoculation (*Munich method*).—Kitt observed during his inoculation experiments, that immunity can be produced in cattle and sheep by a single inoculation of vaccine which had been attenuated by exposure to a temperature of from 185° to 194° F. for 6 hours. This vaccine is about equal in effect to the second vaccine of Arloing, Cornevin and Thomas, and is harmless even if 10 times the ordinary dose be used. The local changes which appear after the inoculation are very slight and hardly noticeable. Kitt obtained a similar attenuated vaccine by means of the steam of boiling water. The results of Kitt's single inoculation are very favourable. In *Bavaria*, in 1894, 3,288 cattle were inoculated (hydrogen vaccine) with but a single loss; in 1895, 4,501 cattle, with pure cultures (4 fatalities); in 1896, 3,446 cattle with Lyons vaccine (14 fatalities); in 1897, 3,659 cattle (hydrogen vaccine), with but 2 fatalities; in 1899, 4,291 cattle (7 fatalities); in 1901, 6,235 cattle (9 fatalities). From 1890 to 1892 in Salzburg, only 5 died out of 4,112 protected cattle, and 71 out of 5,286 non-inoculated cattle during the years 1890 and 1891; in 1894, the single inoculation, with pure cultures, of 1,101 cattle gave the best results (no fatality); in 1895, among 1,071 inoculated with pure cultures there were but 4 deaths; during 1892, in Innsbruck, only 13 out of 4,970 protected cattle; and during 1891 and

1892, in Vorarlberg, 23 out of 8,315 protected animals, and 303 out of 14,835 non-inoculated ones. According to Strebel, the single inoculation with Kitt's dry vaccine disclosed the best immunising qualities; of 34,852 inoculated animals but 0·3 per cent. died of quarter-ill. Only in Lower Austria, the experiments were so far unfavourable in 1893 that 12 animals died of quarter-ill in consequence of the inoculation, on which account the Lyons method was again adopted.

Owing to the difficulty in the preparation of the vaccine from the dried flesh of animals that had suffered from quarter-ill, and the inequality of the preparations made from it, with consequent uncertainty as to the effect, Kitt used the pure cultivations of the bacillus of quarter-ill for the inoculation material, but discarded it for inoculation with a powdered vaccine, prepared from the dried juices of the flesh of quarter-ill, as it is very difficult to procure large quantities of the inoculation matter at the requisite time. (Leclainche and Vallée have recently shown that protective inoculation with pure cultures is effective, sure, and simple.) Finally, Kitt, from researches recently carried out with serum-inoculation, is of opinion that by means of simultaneous or previous serum-inoculation, the danger of accidents from quarter-ill inoculation may be substantially diminished.

Method of Inoculation according to Thomas.—Threads impregnated with lymph and dried "black legine" are introduced into the subcutaneous tissue of the tail, and there allowed to remain for an unlimited period. In America and Algiers, 1,500,000 cattle were, in 1900, permanently immunised by a single inoculation by this method. Schattenfroh recommends repeated subcutaneous injections of culture filtrates (3-10 c.c.) (50 mins. to 170 mins.) at intervals of from 8 to 14 days.

INFLUENZA OF THE HORSE.

Influenza, or pink eye—Diseases of cattle resembling influenza—Influenza of man.

General Remarks on the Meaning of Influenza.—The term, influenza, was originally applied in human medicine to an infective disease with catarrhal implication of various mucous membranes (*la grippe*). The word, which comes from Italian, is etymologically equivalent to "epizoötic" or "distemper." In veterinary medicine it has long served as a collective name for all kinds of equine epizoötics, the respective independence of which could not be proved by the light of the veterinary science of former days. People used the name "influenza" for every feverish, inflammatory malady of one or more of the internal organs which either simultaneously or successively affects a large number of horses. It was known as

equine fever, typhoid fever, typhus, catarrhal fever, and erysipelatous fever. The extraordinary difference of the individual symptoms naturally led to the division of influenza into various forms, such as catarrhal, gastric, pectoral, erysipelatous, typhoid, rheumatic, and bilious influenza.

The first attempt to separate the most important infectious diseases which had passed under the name of influenza, was made by Falke. In his essay, *Influenza of the Horse*, which received a prize from the Royal Academy of Medicine of Brussels, he divided influenza into two principal diseases, which can be diagnostically differentiated with certainty. The first only of these he named influenza. This influenza of Falke is, according to his own description, identical with horse-distemper described later by Dieckerhoff, and with epidemic erysipelas mentioned by Schütz. They are all, however, an infective disease separated from the old collective name "influenza." The other form he names typhus. This typhus of Falke corresponds substantially to the epidemic pleuro-pneumonia of Dieckerhoff.

The correctness of Falke's division has been confirmed by the exhaustive researches of Dieckerhoff, who has demonstrated the fact that by the term influenza there were really denoted two etiologically distinct and well-characterised infective diseases; for which he proposed the two names horse-distemper and epidemic pleuro-pneumonia; while he desires the term influenza to be of merely historical value. Friedberger has, from the first, suggested that Dieckerhoff's horse-distemper should in future be called influenza, as Falke proposed, while the epidemic pleuro-pneumonia should be separately placed. We consider, on the reasons given below, that this view should be upheld, and we accordingly divide the older collective connotation of influenza into the two following independent diseases:

1. *Influenza* in the proper and more restricted sense ("horse-distemper" of Dieckerhoff and epidemic erysipelas of Schütz).
2. *Contagious pleuro-pneumonia.*

Reasons.—1. The infective disease distinguished by Falke as influenza and by Dieckerhoff as horse-distemper, from the early "influenza," is, as an epidemic, historically of more ancient date than contagious pleuro-pneumonia. (See the chapter on the history of both.) It distinctly represents the principal disorder of those epidemic diseases which in earlier times were collectively indicated by the term influenza. On this account, it is desirable, even from the historic standpoint, to reserve the term influenza for it.

2. Exclusion of the term influenza from veterinary pathology would not be easily carried out in practice.

3. The retention appears to us to be demanded in recognition of Falke's work.

4. The term "horse-distemper" indicates nothing in relation to the nature of the disease; nor has it any etymological relationship with the term "influenza"; and for that reason was absolutely foreign to it.

5. The plea that a great number of the German veterinary surgeons united all the items of the older connotations under the name of influenza can so far be said of the term contagious pleuro-pneumonia, as the latter was also originally identified with influenza in the general sense.

History.—According to Falke, influenza was mentioned as early as the time of Charlemagne by his private secretary Eginhard. Dieckerhoff states that it was probably known in the 4th and 5th centuries; in the 14th century, in Italy; in 1648, in Western Germany; and in 1711, in the Eastern provinces of Prussia. Heusinger tells us that it was fully described in 1729 by Löw (*Febris catarrhal, anni 1729 historia*) as an equine epizoötic, which had spread over Italy, Austria, Poland, Wallachia, etc. It is also stated that cases of its having been transmitted to man had occurred. It was observed in 1732 by Gibson and others in London, and in different districts of England, where it raged epizoötically in 1760, 1776, and 1803. The disease was widely disseminated at the end of the 18th and at the beginning of the 19th century, especially during 1805. From that time, influenza has continued to afflict horses in Europe and especially in Germany. The chief epidemics occurred in the years 1813 to 1815, 1825 to 1827, 1836, 1840, 1846, 1851, 1853, 1862, 1870 to 1873, 1881 to 1883, and 1890. Anker, who gave an admirable description of the influenza epizoötic of 1826, in Switzerland, laid stress, even at that time, on the contagiousness of the disease, and stated that in his opinion a volatile infectious matter was the cause. The disease was subsequently described by Waldinger, Körber, Spinola, Hertwig, Hering, Köhne, and others, and in a very explicit manner by Falke in 1862. Influenza spread as an epizoötic in 1872 to 1873 over the greater part of the United States, where it received the name of "pink eye" (French, *Fièvre typhoide*). The last great epizoötic raged in Europe from 1881 to 1883, during which it spread over nearly the whole continent, and flourished chiefly in large towns, generally following the high roads of commerce. For instance, it was present in Berlin over two years, and in Munich for nearly a year and a half. From that time, Dieckerhoff, Schütz, Friedberger, Siedamgrotzky, Lustig, Vogel, and others have ably

investigated and described the disease. This epizoötic appeared in Northern Germany and Denmark during 1890, 1891, and 1892. In the Prussian army, 8,434 horses became affected in 1890; 2,497, in 1891; and 3,645, in 1892. In Copenhagen, 3,000 horses suffered from it in 1890 and 1891. The numbers of cases of the disease in the horses of the Prussian army in the years 1894–1902 included 406, 905, 930, 1,481, 2,066, 861, 325 cases. In recent years a distinct increase of influenza has also taken place.

Etiology.—Influenza, which is as highly infectious as any other disease of horses, can be produced only by infection. The disease can spread in a very short time among a large number of horses. It is transmitted, not in a disconnected and irregular manner, like equine contagious pleuro-pneumonia, but usually by successive infection. The contagium, which, without doubt, is exceedingly volatile, is probably absorbed from the inspired air and is contained in the air expired by affected and convalescent animals, and also in the excrements, as was shown experimentally by Friedberger. It appears to quickly lose its virulence outside the animal body. On the other hand, it seems to be sometimes preserved for a very long time in the animal body. According to the observations of Jensen, Clark and Grimme, stallions which have had the disease may be able to transmit it to mares for months, and even for a year or two, by coition. Dieckerhoff succeeded in transmitting the disease to healthy animals by subcutaneous and intravenous injections of the blood of infected horses; but Friedberger and Arloing failed to do so. Horses are very susceptible to the contagium; their individual susceptibility being independent of age, sex, breed, stable management, feeding, etc. The infection generally takes place directly from horse to horse; though it may be carried by intermediate bearers, such as human beings, litter, harness, thermometers, etc. In many cases, one attack confers lifelong immunity; though a second infection or a relapse not unfrequently occurs. An epizoötic invasion of influenza seems to last for one or two years. Towards the end of its duration, infections become rarer and milder, probably on account of a gradual attenuation of the contagium. At that time, the epizoötic assumes a more sporadic character.

Influenza occurs also in asses and mules. In veterinary literature, we find a few accounts of supposed transmissions of equine influenza to man and dogs (Adam, Walther and Voss).

The period of incubation is, on an average, from 4 to 7 days. According to the observation made in the Prussian army, it was, as a rule, from 3 to 5 days. It sometimes appears, however, to be shorter (according to Blumenberg, in one instance it was only 2 days).

Symptoms.—After a *period of incubation*, the disease appears suddenly, and may attain its highest point of intensity within 24 hours. The organs of circulation, nervous centres, digestive and respiratory mucous membranes, eyes, and subcutis, become especially affected.

1. As *first symptoms*, we usually observe partial or entire oss of appetite, depression, and languor in movement. The internal temperature, which is very high from the commencement, is characterised by a sudden rise up to 108° F. and even more, at which it remains with slight variations for from 3 to 6 days and then falls rather quickly to normal, often within 24 hours. At first, the frequency of the pulse is but little increased in comparison to the height of the temperature, and is usually only from 40 to 50 per minute; but later on it rises to from 60 to 70, and even to from 80 to 100, or more in fatal cases. It generally continues high for some time even after the temperature has fallen. The fever is characterised by unevenness in the distribution of the external temperature of the body. The fact of the internal temperature of apparently healthy horses which are among horses infected with influenza, being high, is of great diagnostic importance. In such infected stables, the temperature of the apparently healthy horses should be taken daily.

2. Great *nervous depression* co-exists with the fever. The animal holds its head down, and appears comatose, as if affected by sleepy staggers. Extreme muscular debility is frequently associated with this stupor. Tremors affect the body and limbs; the hind legs give way when walking; and paralysis of the hind-quarters appears in a few cases. The sclerotic is often of a yellow colour.

3. The *gastric symptoms* form an essential part of the disease. The oral mucous membrane is greatly congested, hot, dry, and often covered with mucus. There is sometimes difficulty in swallowing, when the inflammation of the oral mucous membrane spreads to the pharynx. The animal frequently yawns. Constipation is generally present at the beginning of the disease, and colic may occur. The fæces are formed

into small hard balls and are covered with mucous membranous masses (proctitis). Later on, the constipation often changes into diarrhœa with considerable tenesmus. The fæces are then of a thin, pulpy, and even fluid consistence, and sometimes have a fœtid smell. The peristaltic action of the bowels is usually suppressed, and the hind part of the body is here and there painful on pressure. The urine at the beginning of the attack is alkaline in spite of the high fever, and becomes acid only when the intestinal affection develops. Although it is not often albuminous, it frequently contains large quantities of desquamated epithelium of the bladder. Sometimes the animal stales very frequently (slight catarrhal cystitis).

4. A more or less severe affection of the *eyes* is a rather constant and is a very characteristic symptom of influenza. At first it consists chiefly of a catarrhal, and later on of a phlegmonous conjunctivitis with considerable swelling of the eyelids (chemosis), which may be followed by parenchymatous keratitis and even by an exudative or hæmorrhagic iritis. Generally both eyes become affected. The first symptoms are tears, intolerance of light, great congestion of the mucous membrane of the eyelids, and contraction of the pupil. The eyelids swell considerably, are hot, painful to the touch, and are kept persistently closed. The mucous membrane of the eyelids is glassy, œdematous, and often projects outwards between the eyelids in the form of an orange-coloured protuberance. The conjunctiva scleræ frequently forms a grey, translucent, raised, and tumid ring in the neighbourhood of the cornea. A grey, muco-purulent secretion accumulates between the eyeball and the eyelids, and the eyeball becomes very sensitive to pressure. The cornea at the beginning of the keratitis has a greasy lustre, is iridescent, smoky, and appears as if covered with dust. Later on, a blue or milky opacity forms over its surface. It is considerably injected with blood at its edge; the iris becomes swollen and assumes a yellow colour; and exudate and blood are sometimes seen in the anterior chamber of the eye. Often these inflammatory changes of the eye disappear in a strikingly rapid manner. The deep-seated inflammations of the eye display, exceptionally, a grave and recrudescent character; they sometimes lead to the formation of cataract, and occasionally to blindness from detachment of the retina and opacity of the vitreous humour (Blumenberg, Vanney).

5. During the further process of the disease, *swellings* appear on the extremities, sheath, epigastrium, and lower part of the

chest. We may conclude that these swellings are due to œdema caused by passive congestion; because they are painless, cool, and doughy, and because they appear when the action of the heart becomes weak, during the later stages of the disease. Less frequently, the swellings are of an inflammatory (phlegmonous or erysipelatous) character. The swelling of the extremities causes the gait to be stiff and straddling. The swellings often completely disappear in a very short time on the approach of recovery. Inflammation of the sheaths of the tendons is sometimes observed. In a few cases, an eruption of wheals, outbreaks of sweat, and loss of hair have been seen.

6. The *respiratory mucous membrane* suffers from slight catarrh. At first there is a serous, and, later on, a muco-purulent discharge from the nose; slight swelling of the submaxillary glands; moderate acceleration of respiration; cough; and congestion of the mucous membrane of the vagina. As a rule, the animal becomes a good deal emaciated during the course of the disease; and pregnant mares generally abort.

Course and Complications.—The average duration of the disease is from 6 to 10 days; though severe cases may run on for 2 or 3 weeks, and very mild cases may recover in from 3 to 6 days. As soon as the temperature falls, the appetite usually improves; the peristaltic action of the bowels becomes more brisk; the spirits brighter; the swellings smaller; and recovery becomes complete in from 8 to 14 days. In a few cases, complications set in, especially when the horse has been worked hard during the initial stage of the disease, or during convalescence. The complications are as follows:—

1. *Pneumonia* becomes developed with inflammation of the respiratory mucous membrane, as catarrh of the lungs, with tendency to necrosis of the lungs, or as croupy pneumonia; and may secondarily cause pleuritis. The rate of respiration and of the pulse becomes greatly increased; the temperature rises to a high degree; and the general health suffers considerably. In rare cases, the mucous membrane of the larynx becomes affected with a phlegmonous swelling, which manifests itself by severe dyspnœa and by the production of sounds due to constriction of the lumen of the part.

2. *Cardiac debility*, which is manifested by palpitation of the heart; very frequent, small, and finally imperceptible

pulse; dyspnœa; and passive congestion of the mucous membranes.

3. Grave *cerebral symptoms* (lepto-meningitis and cerebral apoplexy) with unconsciousness and finally paralysis of the brain; spinal paralysis (paralysis of the loins); monophlegia (facial paralysis); and amaurosis (atrophy of the papillæ).

4. Severe *gastro-intestinal inflammation* and *profuse diarrhœa* ending in death.

5. *Laminitis* caused by spreading of the inflammatory swelling of the skin to the sensitive laminæ, or by long standing.

6. *Petechial fever, inflammation of the kidneys, peritonitis, inflammation of the testicles, rupture of the spleen, uncontrollable diarrhœa, tendo-vaginitis, abscesses, muscular spasms, phlebitis,* and *ectropium.*

Anatomical Conditions.—The principal changes of influenza are met with in the *organs of digestion.* The mucous membrane of the pyloric portion of the stomach and intestines (especially of the large intestine, including the rectum) is hyperæmic, swollen, and infiltrated with slight hæmorrhages; and its epithelium is desquamated. The submucosa is yellow and gelatinously infiltrated to a high degree, so that the mucous membrane forms thick, translucent elevations which have a glassy lustre, and which may attain the thickness of about an inch. On being opened they are found to contain a fluid which coagulates in gelatinous lumps. Peyer's patches are enlarged, and present medullary swellings, especially in the neighbourhood of the ileo-cæcal valve. The mucous membrane of the oral cavity and sometimes that of the pharynx show similar changes.

The mucous membrane of the upper *air passages* is catarrhally congested and swollen; and in rare cases the mucous membrane of the larynx is phlegmonously inflamed (gelatinous infiltration of the submucosa), which condition is also found in the subcutis when inflammatory swellings appear on the skin. Schütz states that in the *brain* and *spinal cord* the arachnoideal spaces, particularly on the basilar surface, are filled with a fluid which is generally clear, and which contains leucocytes. The plexuses of the veins may suffer from cloudy swellings. In one case, the lateral ventricles contained nearly $\frac{3}{4}$ oz. of fluid.

The chief *general changes* are as follows: cloudy swelling of the heart, kidneys, liver, spleen, muscles, etc.; slight swelling

of the spleen; small hæmorrhages in the intestines, under the serous membranes, and in the lungs, eyes, and brain; gelatinous infiltration of the renal connective tissue, and mesentery; swelling of the lymph glands; yellow, serous transudations in the cavities of the body; and an imperfectly coagulated condition of the blood.

Prognosis.—Influenza is a mild equine disease. The mortality, which varies at different times and in different places, is on an average from 0·5 to 4 per cent.; being greatest at the beginning of an epizoötic invasion, and least towards its end. Dieckerhoff saw a loss of 4 per cent. in 1,700 infected horses; Aureggio, one of 3 per cent. among 800 horses; Friedberger, one of 9 per cent.; and Siedamgrotzky, one of 10 per cent. The average yearly mortality among the horses of the Prussian army during the years 1886–1893 was only 0·5 per cent. In 1898, but one out of the 2,066 horses which sickened in the Prussian army, died; in 1899–1900, 891 of 4,325 were affected, and but two died. Friis puts the mortality during an epizoötic in Copenhagen (1890) at 1 per cent. It is stated that in 1872 in Philadelphia, 7 per cent. died among 30,000 infected horses.

Differential Diagnosis.—Influenza is distinguished from other infectious diseases by its epizoötic occurrence, rapid dissemination over large tracts of country, great contagiousness, benign character, speedy development, and sudden appearance of the symptoms. It can be separated from equine contagious pleuro-pneumonia by the special implication of the digestive organs and of the eyes; by the severe nervous symptoms; and by the slight participation of the organs of respiration, which in pleuro-pneumonia are the chief seat of the disease from the commencement of the attack. The differentiation between pleuro-pneumonia and influenza can be difficult only at the beginning, when merely general symptoms, such as fever, loss of appetite, and weakness are present. It must be mentioned that these two diseases may simultaneously affect a horse. When the skin is greatly swollen, influenza may somewhat resemble petechial fever, from which it can, however, very soon be distinguished by the absence of petechiæ, by the mildness of its course, and its greater contagiousness.

Therapeutics.—On account of the mild and typical course of influenza, medical treatment is generally superfluous;

varied complications, such as inflammation of the lungs, inflammation of the brain, myocarditis, and other sequelæ. Pfeiffer states that the disease can be transmitted by inoculation to monkeys and rabbits. We have no proof that human influenza is identical with equine influenza. It is even improbable that the two diseases are in any way connected together. In our opinion they are entirely different infective diseases, which are in no respect etiologically related to one another. The respective differences between the course of the outbreaks, the morbid phenomena, and the fact that the transmission of the virus from man to horse, or *vice versâ*, has never been proved to have taken place, are strong arguments against the identity of the two diseases. The same remark holds good with respect to any supposed identity between human influenza and canine distemper.

CONTAGIOUS PLEURO-PNEUMONIA OF THE HORSE.

(*Pleuro-pneumonia Contagiosa.*)

Nature, Occurrence, and Pathogenesis.—Pleuro-pneumonia is an infective inflammation of the lungs, in the course of which the pleuræ (pleuro-pneumonia), and large organs of the body, such as the heart and kidneys, participate in the morbid process. Its infective matter has not yet been found with certainty. The respective statements of bacteriologists, to which we shall refer later on, differ greatly from each other. The disease occurs enzoötically, principally in places where large numbers of horses are kept; for instance, in cavalry regiments, army depôts, breeding establishments, and stables of horse dealers; and is more frequent in towns than in the country. Horses from 5 to 10 years appear to be more susceptible to it than older horses, some of which, perhaps, have already had the disease. One attack generally gives immunity for several years, and sometimes even for life, although exceptions are not uncommon.

Horses are not so susceptible to pleuro-pneumonia as to influenza. Rust states that in a thoroughly infected regiment of 705 horses, only 245 took the disease; 400 proved to be immune, and 60 had had the disease. Horses in good health are less susceptible than those with a weak constitution. Over-exertion, chill, catarrh of the lungs, and bad ventilation in the stable are pre-disposing agents. Consequently the disease is, as a rule, more frequent in winter than in summer.

The contagium can be directly transmitted from animal

to animal by the respired air. It can, for instance, be introduced by remounts and working horses, and is transmitted from horse to horse. The infection, however, is carried usually by intermediate bearers, such as human beings (especially veterinary surgeons and grooms), healthy horses, dung, clothes, forage, strange stables, and even dogs. The infected horses of the civil population of garrison towns are a continual source of infection for army horses. Convalescent horses are particularly dangerous ; because they can carry about in their bodies the infectious matter, frequently, as it seems, for weeks (dead lung-deposits). It has not yet been positively proved whether a transmission of the disease, without direct or indirect infection, by means of a miasma, that is, by a spontaneous development of the contagium outside the animal body, may occur. The frequency of pleuro-pneumonia in badly ventilated and insufficiently drained stables, and the fact that not uncommonly sporadic cases may be observed in a stable throughout the whole year, have led to the supposition that the infective matter may be preserved in a stable as a stable-miasma for a long time, and may thus cause the disease to become stationary. On the other hand, the occurrence of such cases may be explained by the supposition that a direct or indirect infection is transmitted by one or more infected horses which are slowly recovering from the disease, and which are apparently healthy ; or by direct or indirect infection from outside, which is always possible, considering the wide dissemination of the disease. The question mooted by Peters, of the connection between pleuro-pneumonia and variations of surface-water, requires to be further investigated. The appearance of pleuro-pneumonia in a stable is different from that of influenza, which attacks horses in turn. The order in pleuro-pneumonia, on the contrary, is quite arbitrary, as if it was regulated by intermediate bearers. The army horses which occupy corner stalls are most frequently and most severely attacked, probably on account of inferior ventilation.

The course of the disease is generally most rapid at the commencement of an outbreak. A case has been known of 42 horses of one squadron becoming affected within 2 days, and another case of the disease spreading within 3 days through an entire military stable. Frequently the horses of a whole regiment become infected within a fortnight. On an average, the epizoötic disappears 5 or 6 weeks after the occurrence of the last case, provided that no fresh case

is introduced. Subsequently, the course of the disease generally becomes chronic and indolent, and may remain stationary in some stables, if the horses in them have become chronically affected with lung disease (cavities of the lungs), and, on that account, maintain the supply of the infection; or the disease may be introduced again and again from outside by apparently healthy horses (in the army, especially by remounts).

The respiratory organs are the chief places of entrance of the contagium, which probably gets first into the lungs by inspiration, and from thence into the blood. It is not improbable that the infection may also take place from the digestive canal.

The period of incubation is generally given as from 5 to 10 days; although frequently it may be only from 1 to 4 days, or may sometimes extend to 10 days or a fortnight. It is very difficult to fix its exact duration; because it is impossible to find out the precise time the infection takes place. Consequently, many of the statements made as to its length are contradictory. In no case should we, as was frequently done, calculate the period from the time the healthy horse has been placed in an infected stable to the appearance of the first symptom. We need hardly say that the period of incubation is that interval which lies between the absorption of the infective matter into the body and the appearance of the first symptom. The time at which the absorption of the contagium takes place cannot, as a rule, be determined.

Bacteriology.—1. Klebs, Ebert, Koch, Friedländer, Fränkel, Zäslein, Salvioli, and others have found micro-organisms, which are the exciters of inflammation in human lungs, in the form of streptococci, and often diplococci, and which are surrounded with a capsule.

2. In cases of inflammation of the lungs of horses, Peterlein, Perroncito, Brazzola, and Mendelsohn were the first to find in the inflamed parts of the lungs streptococci, the cocci of which were separated from each other by a bright zone; and diplococci (Peterlein). Perroncito describes as "*bacterium pneumoniæ crouposæ equi*," large cocci which are almost spherical or ovoid, single or in twos (diplococci), sometimes also in threes, and rarely connected in chains (streptococci), and which are often surrounded by a bright, gelatinous ring. Perroncito distinguishes these cocci from those of man by the circumstance that they are always pathogenic in guinea-pigs and rabbits, and that they cannot be stained with the solutions which are used for the staining of the capsule of the cocci of man. In all these experiments the pathogenic character of the cocci in question has been proved neither by pure cultivations nor by inoculation.

3. Lustig prepared in 1885 pure cultures of a micro-organism which he considers to be the contagium of equine contagious pleuro-pneumonia,

and with which he made inoculations later on. He obtained in his bacteriological researches six different cultivations, of which he considers the sixth, a yellow cultivation, to be the exciter of this disease. The cultivation grows chiefly on the surface, but only to a small extent, and also grows in the track and at the bottom of the puncture in gelatine. These stab-cultivations have almost the form of a nail. The cultivations consist of very minute, ovoid bacilli, which can be stained with Gram's solution. They are stained easiest in a saturated solution of dahlia. Lustig obtained the following results from inoculations with these cultivations: 9 horses that were inoculated in the thoracic cavity became affected with pleuro-pneumonia, with the result that 2 died and 7 recovered. No. 6 culture was able to be obtained out of the pleuritic exudate in one of the horses while it was alive, but which subsequently died. Four horses which had been inoculated in the thoracic cavity were afterwards exposed with five horses which had not been similarly inoculated, to the natural infection, with the result that the former remained healthy and all of the latter became attacked by the disease. Schütz and Lüpke have not been able to find the micro-organism observed by Lustig, and have declared that they do not consider it to be specific.

4. Schütz believes that the contagium of equine pleuro-pneumonia consists of small oval bacteria which are usually grouped together as diplo-bacteria, and which have been formed by the transverse division of individual bacteria. These bacteria can be stained with methylene blue and all basic aniline colours, and are characterised by the fact that they become decolourised when Gram's method is used. We can sometimes recognise around them a ring which in some cases can be stained, but cannot in others. Stab-cultures on meat-water-peptone-gelatine produce small, white, spherical tufts which do not liquefy the gelatine and which do not spread over its surface. Cultivations in broth show white flakes at the bottom of the tube, and on agar-agar they form small, opaque, grey tufts. The pure cultivations can be transmitted to mice, rabbits, pigeons, and guinea-pigs, but not to pigs or fowl. The inoculation produces in mice a septicæmia which generally ends in death in from 24 to 48 hours. According to Schütz, the cultivations can be transmitted effectively to horses, and when injected directly into the lungs by means of a Pravaz's syringe they produce contagious pleuro-pneumonia, which has the same symptoms and course as those observed in natural cases of this disease. The essential changes shown on *post-mortem* examination were multiple gangrenous patches in the lungs with parenchymatous degeneration of the most important organs. The characteristic bacteria were found in the body of the inoculated animals. According to Schütz, bacteria of contagious pleuro-pneumonia are found most numerously in the lungs and in the exuded matter of the pleuræ, and are also met with in the nasal discharge and in expired air (Rust). The development of the bacteria ceases at a temperature of 50° F., and evidently is favoured by moist heat, such as that of a manure heap. Nothing positive is known respecting the duration of life of the bacteria outside the animal body. They are usually supposed not to be able to live longer than 6 weeks in the animal body. The contagium may, however, continue active for a very long time, especially in encysted deposits in the lungs.

Baumgarten, Hell, Foth, Tröster, and others oppose the view that Schütz's bacteria are specific, while Rust and Fiedler support it. Baumgarten considers that this subject is not fully explained. Hell maintains that with our present means of investigation, the bacteria of contagious pleuro-pneumonia cannot be differentiated from those of the streptococci of pus, and from the streptococci of erysipelas. In fact, Hell believes that the ovoid cocci and diplococci of Schütz have a pathogenic effect in the infection of horses with pleuro-pneumonia; but as we have no positive proof of their being specific, he maintains that we are justified in supposing that these ubiquitous micro-organisms have only an injurious influence on the course of pleuro-pneumonia, and that they contribute to the production of the secondary affections, especially of the necrotic foci of the pleuritis. Hell further states that protective inoculation with Schütz's bacteria, which at first promised good results, has not fulfilled the expectations formed of it. Fiedler, on the other hand, has obtained the same bacteriological results and has arrived at the same conclusions as Schütz. He also states that he has experimentally produced pleuro-pneumonia in a horse by inoculation of cultivations of these bacteria.

5. Lignières has described the disease as an equine Pasteurellosis produced by a bacterium (cocco-bacillus) of the Pasteurella group, and identical with the Argentine Pasteurellosis of cattle. His view is that every case of pneumonia in horses is really Pasteurellosis. When the cocco-bacillus is very virulent, influenza is said to develop. In the case of pleuro-pneumonia, the cocco-bacillus is associated with the streptococcus of strangles (Schütz's bacteria); and its predominance ultimately asserts itself.

It therefore appears that the bacteriology of equine contagious pleuro-pneumonia is still an open question.

Anatomical Changes.—In the lobular form of pleuro-pneumonia, which furnishes the largest number of subjects for a *post-mortem* examination, we find a multiple hæmorrhagic, gangrenous pneumonia with secondary pleuritis and parenchymatous degeneration of the chief organs of the body.

1. We find portions of the *lungs*, of greater or less size, thickened, hepatised, and devoid of air, especially near the root of the lungs and in the lower sections; and bright foci, which are distinctly defined from the neighbouring tissues, sprinkled over the dark-red sections of the hepatised parts. Usually, several of these foci are present, though sometimes only one may be met with. They vary in size from a pin to a child's head. At first they are very small; of a greyish-red colour (hæmorrhagic foci), and are surrounded by a whitish zone of emigrated leucocytes (limited reactionary inflammation).* Later on, they become yellowish, resemble tinder in appearance (dry necrosis), and then take the form of cavities, which may be as small as a

* Inflammation which is caused by reaction to external influences, and which leads to demarcation or casting off of dead portions of tissue.—TR.

pea or as large as a hen's egg, and which contain necrotic lung tissue. These cavities are surrounded by a smooth capsule. There are other foci which contain greasy, fœtid, watery pus (gangrene of the lungs), by reason of the necrotic parts of the lung undergoing watery disintegration, in consequence of the admittance of air. We also find in the lungs suppurating foci, which are generally of rather a large size, and which contain a whitish pus, mixed with dead lung tissue (suppurative demarcation of the necrotic parts). It may often happen that none of the foci which we have just described are to be seen in the lungs; although, during life, distinct symptoms of such a localised affection may have been present. In these cases, we must assume that absorption of the necrotic tissue has occurred. The remaining tissue of the lungs is, more or less, hyperæmic and œdematous.

2. The *pleuræ* show signs of a diffuse, exudative pleuritis, the starting-point of which, in the large majority of cases, is from necrotic deposits which are situated on the periphery of the lungs. This pleuritis may, however, appear without these foci being present. The contents of a necrotic deposit in the lungs rarely breaks through into the pleural cavity. Both the visceral and costal layers of the pleuræ are congested diffusely and in spots, and are infiltrated with hæmorrhages; are dull and rough; and are often covered with soft, red granulations. Over them we find soft, yellow layers, which are partly membranous and partly coagulated in a reticular manner, and which can usually be easily removed. The cavities of the pleuræ generally contain a considerable quantity of fluid, which may amount to 7 gallons or more. It is usually turbid; is of an orange, grey-red, brown-red, or dirty-greyish colour; and is generally mixed with numerous yellow flakes which form a sediment when the fluid has been left to stand in a glass. The pleuritic exudate sometimes consists of pure pus (empyema), and less frequently of blood (hæmatothorax). Pneumothorax is very rarely present. The exudate compresses the lungs and pushes them away from the thoracic walls. In consequence of the exuded fluid becoming organised, the lungs become adherent to the costal walls and diaphragm, and fibrous, villous growths develop on the pleuræ.

3. The *other organs of the body* are in a state of parenchymatous inflammation and fatty degeneration. The ***muscular tissue of the heart*** is, as a rule, brownish-grey in colour, soft, and suffers from cloudy swelling. In severe cases it shows well-

marked fatty degeneration, is of a clay colour, and is occasionally infiltrated with a large number of small, yellowish-white foci. The *liver* is enlarged, of a clay colour or sometimes icteric, and presents signs of fatty degeneration. The *spleen* is flaccid, and its pulp increased and often infiltrated with hæmorrhages. The *kidneys* are swollen, brittle, and sometimes show numerous hæmorrhagic foci. The *lymph glands*, especially the bronchial and mediastinal glands, are enlarged, softened, and exhibit a grey-red colour on section. The *muscles of the body* are soft, and of a yellowish-brown colour. We may frequently find small *hæmorrhages* under the serous membranes, and slight endocarditis. The *blood* suffers from less change than any of the tissues.

4. The *mucous membrane of the stomach and intestines* is frequently hyperæmic, swollen, infiltrated with hæmorrhages, and sometimes even ulcerated. Its epithelium is desquamated, and its lymphatic glands are swollen, and often fall out. The bronchial mucous membrane is also swollen and inflamed.

The anatomical changes of the less frequent lobar form are those of croupous pneumonia.

Symptoms.—The symptoms are of such a varied character that it is quite impossible to give an exact description of them. We shall first mention those most frequently observed, and then put the complications under a separate heading. Pleuro-pneumonia appears rather suddenly, or is preceded by premonitory symptoms, such as general faintness, loss of appetite,* cough, etc. The first regular symptom is a rapidly increasing temperature, which generally rises to 104° or 106° F., and which frequently begins with rigors. It often rises 6° F. within a few hours. The rate of the pulse increases to from 50 to 70 beats a minute, and often, later on, to 80 or 100, or even more. On the whole, the pulse is more frequent in pleuro-pneumonia than in influenza. At the same time, during the later stages of the former disease, it is very small, and there is severe palpitation of the heart. We have also depression and muscular weakness, though not to such a high degree as in influenza; and the conjunctiva and other visible mucous membranes are congested, and are deeply tinged with yellow. There is almost entire loss of appetite. The character of the lung affection may vary in cases of equine contagious pleuro-pneumonia, which presents the two following entirely different clinical aspects.

* McFadyean remarks that, contrary to the usual opinion, the appetite, as a rule, is fairly well sustained in this disease.

1. A *lobular* inflammation of the lungs with tendency to gangrene of the lungs and pleuritis.

2. A *lobar* inflammation of the lungs, which is of a mild character, and which has no tendency to gangrene and pleuritis.

(1) Lobular-pneumonic form of Equine Pleuro-pneumonia.

This affection of the lungs is localised and manifests itself at first by cough, and accelerated and difficult breathing, as well as by high fever and other general symptoms. If the pneumonic foci are very small, no dulness of the lungs will be found on percussion, and auscultation will reveal nothing abnormal beyond the fact that the vesicular breathing is strong and shrill. Pleuro-pneumonia in such cases is difficult to diagnose, and may be easily mistaken for influenza. If several pneumonic deposits be close together, lobular, irregular dulness may often be found on percussion, especially in the antero-inferior parts of the lungs, such as directly behind the shoulder, and particularly on the left side near the heart; and weakness of the vesicular respiratory murmur. The presence of this lobular pneumonia can generally be ascertained after the third day of the attack. At the same time we may observe the appearance of a rusty-yellow nasal discharge, which sometimes dries into a brownish-yellow crust near the nostrils. The cough becomes weaker and the difficulty of breathing increases.

The extension of the inflammatory process to the pleuræ is at first manifested by the dry friction sound, and by the highly sensitive condition of the affected places on the thoracic walls. Respiration becomes abdominal. With the accumulation of a pleuritic exudate in the thoracic cavity, the dulness becomes more extensive; it assumes a marked horizontal direction; and may extend over two-thirds or more of the thoracic cavity. On auscultation in such cases, we find that there is no vesicular respiratory sound, or that there is bronchial respiration on the boundary of the dulness. The change of lobular-pneumonic places into necrotic cavities is shown externally on percussion by a tympanitic sound, or by a cracked-pot sound (*olla rupta*). The same sounds are heard with localised pleuritic exudates. With the increase of the pleuritic exudate, respiration becomes extremely difficult, and the horse while trying to breathe, widely distends his nostrils, which, so to speak, take the form of trumpets. The course of the fever is sometimes typical; at other times, atypical.

If the disease runs a favourable course, improvement takes place after the above described symptoms have lasted for 6 or 8 days. This improvement is generally preceded by a critical polyuria and rapid lowering of the temperature. The recovery is complete after a period of convalescence, which lasts from 2 to 4 weeks. Such a rapid and favourable course can be expected only when the animal has not suffered from pleuritis with abundant exudation. In severe cases, the animal may die during the first week. Death is caused chiefly by a series of grave complications.

(2) Lobar-pneumonic form of Equine Pleuro-pneumonia.

This, which is the milder form of pleuro-pneumonia, is characterised by its typical course and large areas of dulness. Exudative pleuritis is rare in it. The inflammation of the lungs begins also on the antero-inferior parts of the lungs, and extends rapidly over large sections of these organs. In the majority of cases the inflammation is unilateral; that of the left side occurring about twice as often as that of the right side. Friedberger found only 8 patients afflicted with bilateral pneumonia out of 70 cases which he had examined in order to decide this point. In the initial stage, percussion of the thorax produces a tympanitic sound, which is seldom quite pure, and is generally somewhat dull. The second stage, namely, that of hepatisation, is distinguished by a dull percussion sound with the feeling of considerable resistance to the hammer. Tympanitic sounds are also noticed on the boundary of the dull sound. In the third stage, we again find the tympanitic sound. This typical character of the percussion sound is not met with in abortive cases; because, owing to the shortness of the course of the disease, percussion reveals no changes, or only trifling ones. At the beginning of the attack, auscultation of the lungs, and especially of the boundary zones, reveals crackling râles; in the second stage we hear, chiefly during expiration, bronchial breathing; and in the third stage, moist râles. Auscultation of the healthy parts of the lungs manifests increased and frequently a rough vesicular respiratory murmur.

The respiration, which is generally between 20 and 60 a minute, is accelerated and difficult, in proportion to the height of the fever and to the extent of the inflammation of the lungs; and is chiefly costal. The nostrils are widely dilated and sometimes give out a peculiar flapping sound, which Dieckerhoff

believes to be due to temporary adhesion of the mucous membrane of the false nostril to that of the opposite side. The expired air is abnormally hot. The sufferer either persistently stands up with the fore legs as a rule wide apart, or lies down only on the affected side, and is careful to avoid any movement. We may hear at the same time a shallow, short, and painful cough, which is often entirely suppressed. At the beginning of the attack, in about 25 per cent. of all the cases, especially in the severe ones, and in those in which the pneumonia assumes a well-marked hæmorrhagic character, we find a nasal discharge which may vary in colour from saffron-yellow to dirty-yellow, and which, on drying, forms yellow crusts about the nostrils.

The ordinary course of lobar pleuro-pneumonia is typical in that the stages of engorgement, hepatisation, and absorption, follow each other at equal intervals of time. The course of the abnormally high temperature is also typical. The disease usually reaches its crisis on the fifth or sixth day, after which resolution begins. The dulness often disappears in a surprisingly short time; rhonchi take the place of bronchial respiration; the breathing becomes more tranquil; and the cough looser. Recovery, as a rule, takes place in from 8 to 14 days.

Complications of Contagious Pleuro-pneumonia of the Horse.—During the course of this specific pleuro pneumonia, besides lobular or lobar pneumonia and pleuritis, we may have a series of symptoms which, with a combination of pneumonia and pleuritis or by themselves, may present the aspect of the disease we are considering. The heart, membranes of the brain, and kidneys are sometimes so exclusively the seat of the disease, that the lungs may be unaffected. According to the statistics collected on one occasion, inflammation of the lungs and pleuræ was absent in 27 cases out of 117. The complications are as follows :—

1. A severe *cardiac affection* shown by parenchymatous inflammation of the myocardium, which is one of the most common and most dangerous complications of pleuro-pneumonia, and which manifests itself by a very frequent and small pulse, that finally becomes imperceptible; palpitation of the heart; extreme weakness, and general symptoms of congestion, especially in the lungs (dyspnœa).

2. Grave *gastric symptoms*, such as severe colic, diarrhœa, and bloody fæces.

3. A *hæmorrhagic or catarrhal (parenchymatous) nephritis*,

which is characterised by hæmaturia ; presence of casts of blood and of exudates in the urine ; great weakness of the hind legs ; anasarca (parenchymatous nephritis) ; and increasing general anæmia. We have seen fatal cases of this complication, in which the inflammatory lesions in the lungs had completely healed.

4. *Septicæmia* and *pyæmia* following the formation of serous and suppurating foci in the lungs.

5. *Cerebral convulsions* and *symptoms of a leptomeningitis* in consequence of the contagium of pleuro-pneumonia attacking the cerebral membranes (the so-called nervous form of pleuro-pneumonia). These cerebral symptoms are sometimes the first and only symptoms of pleuro-pneumonia. As a rule, death rapidly ensues from paralysis of the brain ; in rare cases, staggers is developed. A wholly unexpected recovery may occasionally take place.

During the progress of the disease, we may also observe copious hæmorrhage of the lungs ; hæmorrhage of the kidneys ; intermitting inflammation of the tendons and their sheaths ; laminitis ; internal inflammation of the eyes (iritis exudativa et hæmorrhagica) ; roaring (frequently in 10 per cent. of the affected horses) ; epileptiform convulsions ; muscular spasm in the region of the facialis and trigeminus ; muscular rheumatism ; arthritis ; rupture of the spleen ; weakness of the loins ; paralysis in the region of the ischiadicus ; paralysis of the rectum, tail, bladder, and penis (in very rare cases) ; chronic endocarditis with valvular lesions ; chronic pericarditis ; broken wind ; anasarca (a symptom of parenchymatous nephritis) ; pharyngitis ; phthisis pulmonum ; general mal-nutrition ; general falling out of the hair ; and paralysis of the pharynx. During the course or towards the end of an attack of pleuro-pneumonia, the affected animal may become seized with influenza, strangles, or petechial fever, in which case we have so-called mixed infection. Malkmus has published a communication on occasional cases of recovery of cancer of the hoof in the course of contagious pleuro-pneumonia.

Course.—The course of equine pleuro-pneumonia is exceedingly irregular, and may be typical, atypical, complicated, acute, chronic, or, not uncommonly, abortive.

1. A *typical course* is seen in horses which have a strong constitution, and which are affected either with the lobar form of pneumonia, or with a slight lobular affection of the lungs. The high temperature remains steady (*febris continua*) for from 5

to 8 days, after which it gradually falls, and the pneumonic or pleuritic symptoms disappear. Such cases usually end favourably; although the period of convalescence is longer (2 or 3 weeks) than that of influenza. Residence in a dry and well-ventilated stable, or in the open air, favourably influences the course of the disease. As soon as the first symptoms of the disease appear, the animal should at once be relieved from work, and should be carefully nursed.

2. *Abortive attacks* are usually met with during mild outbreaks and under favourable external conditions. The disease then lasts only for a few days and assumes the character merely of an infectious bronchial catarrh. These epizoötic catarrhs of the respiratory mucous membranes generally precede the severer forms of pleuro-pneumonia. We find occasionally only premonitory symptoms of this disease. Sometimes, elevation of temperature is the only symptom present.

3. An *irregular course* with tendency to complications is generally found in weak animals; in those kept in unsanitary stables; in those which have been worked in spite of evident illness; and in horses suffering from gangrene of the lungs.

4. The *chronic course* of equine pleuro-pneumonia is attended by very serious consequences, and results from necrotic parts of the lungs becoming caseated and encapsuled, as in bovine pleuro-pneumonia. The contagium may remain virulent for months in these encapsuled foci. Generally, the only outward signs shown by the animal are loss of condition and inability to bear fatigue. Some horses become broken-winded, and others exhibit no marked sign of ill-health. Such chronic cases are a dangerous source of infection, if the encysted deposits communicate with a bronchus. The presence of such deposits may sometimes produce in the affected horse an acute and even fatal illness, after apparent recovery from the primary disease, as was shown in a case observed by Schwarznecker. In this instance, a 13-year-old cavalry horse contracted fatal inflammation of the lungs and pleuræ after a heavy field-day, 3 months subsequent to the disappearance of pleuro-pneumonia out of the affected squadron. This horse had remained healthy up to that time, and his temperature, which was taken daily, was always found to be normal. *Post-mortem* examination revealed in his lungs an old, encapsuled, caseous nodule, which, on account of the violent exertion, had burst into the thoracic cavity and had produced, there, an acute sero-fibrinous form of equine contagious pleuro-pneumonia.

Diagnosis.—The diagnosis of this disease is sometimes very difficult, on account of the not uncommon occurrence of abortive cases in which there are no pneumonic symptoms. Taking the temperature of the apparently healthy horses twice daily, is often the only means for the early recognition of the disease, when it breaks out among large numbers of horses which are kept together. This use of the clinical thermometer has proved of great service, especially in the army. When dealing with large numbers of horses, it is well, for prophylactic reasons, to regard as contagious pleuro-pneumonia, any feverish acute disease which shows lung symptoms, if it occurs without any assignable cause. We should, of course, exclude pneumonia due to chill, foreign bodies going the "wrong way," metastasis, etc.

Prognosis.—The mortality of pleuro-pneumonia varies at different times. It depends on the progress of the epizoötic, constitution of the animal, character of the nursing, amount of the contagium absorbed, and nature of the pneumonia. The course is generally favourable under well-regulated dietetic and hygienic conditions, and when the infected horse is well nursed. It has been observed, especially in the army, that the fact of withdrawing the affected horse at once from work and placing him in the open, has a good influence on the disease. The mortality, which varies from 20 per cent. to *nil*, is higher at the beginning of an epizoötic than subsequently. During 1886 to 1893, over 17,000 horses suffering from pleuro-pneumonia, were treated in the Prussian army, with a loss of about 4 per cent. The proportion was the same in Bavaria. According to the observations of Dieckerhoff, the loss from this disease amounts to 15 per cent. In any case, the prognosis of pleuro-pneumonia is on the whole not so hopeful as that of influenza. The fact that the percentage of deaths is almost equalled by the percentage of cases in which pleuro-pneumonia leaves behind chronic disturbances of breathing after apparent recovery, makes the prognosis still more unfavourable. In connection with the necrotic disintegration of the lung tissue, we often find, in addition to the encapsuled necrotic deposits, the development of chronic intestinal inflammation, which leads to thickening and induration of the parenchyma of the lungs, with consequent diminution of the respiratory surface of the lungs. Chronic inflammation of the mucous membrane of the bronchi, with formation of bronchiectasis and peribronchitic deposits, are also met with. The exudative pleuritis not uncommonly gives rise

to adhesions of the lungs with the thoracic wall or diaphragm, and in other cases to chronic hydrothorax and empyema. All these conditions cause chronic dyspnœa and decreased capacity for work, and thus form a not uncommon cause of "broken wind." To this must be added the other complications of contagious pleuro-pneumonia, such as roaring, inflammation of the tendons, paralysis, etc., which lead to chronic unsoundness.

Therapeutics.—The antipyretic treatment of pleuro-pneumonia, similar to that of influenza and of all other infective diseases, has not answered expectations; for its action has been found to be neither permanent nor very effective. For reducing temperature, we have generally to content ourselves with cold packing and enemas of cold water. Antipyretic remedies are necessary only when the elevation of temperature is very great, and long continued, or when there is a dangerous cardiac weakness. In such a case, we recommend antifebrin (doses of 1 oz. every 3 hours), digitalis, alcohol, and camphor. In a great number of cases we have used digitalis with very good results, but only in large doses. We give on an average 2½ to 3 drams of the leaves in a single dose. Instead of digitalis we may give tinctura strophanthi, 3 to 7 drams internally, but not subcutaneously. We may give with advantage alcohol, the best form of which is wine (1¾ to 3½ pints *pro dosi*); and camphor subcutaneously as spirits of camphor, or oil of camphor, 3 to 7 drams each. The following are useful cardiac stimulants: caffeine (1⅓ drams caffeinum natrio-salicylicum dissolved in 3 drams of water, subcutaneously), hyoscine (⅙ grain), and atropine (¾ to 1½ grains).

Priessnitz bandages and other counter-irritants are commonly used, and generally with good results in inflammation of the organs of the chest. We may also stimulate the activity of the skin by frictions with oil of turpentine (1 to 10 of spirits of camphor). One of the best blisters is oil of mustard diluted with alcohol (1 to 12–20). It has been strongly recommended by Dieckerhoff, and may be applied several times in case of need. Cantharides ointment and croton oil should not be used. For the removal of the inflammatory products from the lungs, we may give expectorants, such as ammonium chloride, alkalies, and compounds of antimony; and to promote the absorption of the pleuritic exudate, we may employ diuretics and absorbent remedies, such as digitalis, alkalies, fructus juniperi, potass. acetas, and pilocarpin in doses of 1½ to 3 grains, or

50 grammes (1$\frac{2}{3}$ ozs.) subcutaneously, carried out in the 10th Dragoon regiment, estimated the protection afforded at so low a standard that, in a strict sense, a protective inoculation could not exist. According to Weiszhaupt, fresh attacks of pleuro-pneumonia occurred in spite of inoculations; and, consequently, inoculations were again abandoned as useless.

In 1898, Töpper reported on his experiments with blood-serum inoculations, as a result of which horses were to be made immune in from 6 to 8 weeks. He laid stress on the fact that the epidemic prevailed in groups of horses mostly for a period of from 4 to 6 weeks, and that it accordingly subsided always directly after inoculation. The inoculated blood-serum must not be less than 150 grammes (5 ozs.) in quantity, and must be taken from horses of the same stable (or depôt), which had already passed through the disease, and had been free from fever for at least from 3 to 6 weeks. Schwarznecker, having regard to the fact that Töpper always began the inoculations for the first time, 4 to 6 weeks after the outbreak of the epidemic, holds it as very questionable whether the subsidence of the disease is really attributable to the inoculation, and not to the natural course of the epidemic. In many groups of horses, according to Schwarznecker, the duration of the epidemic has mostly been from 2 to 6 weeks, after which it subsided of itself. With regard to protective inoculation with blood-serum, it was stated in the Prussian Military Gazette, in 1898, that it had no practical value. The duration of the epidemic was shorter among those groups of horses where no inoculation was tried than in those in which it had been fully carried out. Peschke inoculated 18 horses, after Töpper's method, without any result; indeed, 15 of the number contracted pleuro-pneumonia, two of these very seriously. According to Peschke, inoculation is not merely ineffectual, but is even attended by great danger to the inoculated horses. Matthias states that serum inoculation can have no appreciable effect in bringing a stationary epidemic of pleuro-pneumonia to a conclusion, as the immunity produced by the inoculation lasts but about 6 weeks.

In 1899, the majority of the military reports expressed an unfavourable opinion as to the value of serum inoculation. As in the preceding year, the duration of the epidemic, in those sections of the cavalry where inoculation had been carried out, was, on the contrary, more prolonged.

In 1900, serum inoculation not only failed to give positive results, but was even followed by distinct sequelæ; for example,

among 500 inoculated horses of one regiment, there followed extensive swellings and abscesses at the seat of inoculation, so that many were not fit for service for 14 days. Some horses actually died of malignant inoculation-œdema.

Grammlich published the results of serum-inoculation by Töpper's method (1898) carried out in three regiments of lancers of the Guard. The course of the epidemic was sensibly shortened by the inoculation; absolute protection was not, however, attained; for an inoculated horse sickened 19 days after the inoculation.

Tröster, in accordance with the instructions of the Prussian War Department, carried out serum-inoculation in the winter of 1898–9 in 17 batteries and 6 squadrons; 50 grams (1⅔ ozs.) of serum being used for each horse. The inoculation caused a stoppage of from 5 to 36 days in the course of the epidemic; but was, however, not lasting, as the epidemic did not disappear after the inoculation. This short-lived protection through inoculation can only be of use, according to Tröster, when the object is to arrest for a couple of days, the outbreak among a great number of horses. The experimental efforts at curing horses suffering from pleuro-pneumonia by means of serum-inoculation have had no result. In 1899 Tröster carried out new experiments with larger quantities of serum (500 ccm. (16⅔ ozs.) injected into the breast, in two portions) on 58 horses. The period of protection lasted about 40 days. The limited data obtained appear to Tröster to warrant the conclusion that a prolonged protection may be procured by the use of a dose sufficiently large, while not so great as to involve danger from the inoculation. It is, however, very difficult to obtain large quantities of the inoculation matter, and, therefore, the use of the inoculation method is substantially restricted. For example, for inoculation of 60 horses in a squadron, the blood of 15 convalescent ones is necessary. In order to arrive at this number the epidemic must already have prevailed for a considerable time. The depôt of horses would by then have become so far plague-swept that a successful campaign against the contagious pneumonia would be hopeless.

The series of inoculations carried out by Tröster—in the summer of 1900 (784 horses, 150 grams (5 oz.) of serum), and in the winter of 1901 (635 horses, 100–150 grams (3⅓ oz. to 5 oz.) of serum; later, a second inoculation with 150 grams [5 oz.])—were only partially successful. The collective result obtained by him, from the inoculation of about 3,000 horses, was that a period

of protection of no longer duration than about three weeks by inoculation could be obtained.

In 1900, Mieckley inoculated 200 horses in the Bekerbeck stud with 200 grams (about 6¾ oz.) of serum each. The result was negative; in spite of the inoculation, 113 horses sickened of contagious pleuro-pneumonia, and 14 of them died.

In Denmark, according to Friis, 546 horses were inoculated, of which 26 sickened after the inoculation (150 grams (5 oz.) of serum=efficient minimal dose); the inoculation results were regarded as favourable. In Italy, Della Noce and Giancola profess to have obtained favourable results from serum-inoculation.

Skalma.—Under this name Dieckerhoff describes several forms of equine disease, the chief symptoms of which are an irritable cough; nasal discharge and distressed breathing without percussion or auscultation revealing any morbid change; high temperature; loss of strength; and anæmia of the mucous membranes. The appetite remains comparatively good, and there is a rather long period of convalescence, which lasts usually 3 or 4 weeks. According to this, the disease may be regarded, on the whole, as an epizoötic bronchitis which has a subacute course. It is supposed to be sometimes complicated with inflammatory irritation of the pharyngeal mucous membrane; and in severe cases with a diffuse exudative inflammation of the pleuræ (!). The course of the disease is generally mild. The disease appears in the form of a stable enzoötic which may remain 2 or 3 months, or even longer, in a stable, and is said to be directly infectious. Dieckerhoff believes that "skalma" is one of the diseases which, like equine contagious pleuro-pneumonia and "equine distemper," were formerly included under the heading of "influenza."

We cannot acknowledge that skalma is a clinical unit. Some of Dieckerhoff's cases of skalma may have been closely related to pernicious anæmia. Its epizoötic character, the well marked anæmia of the mucous membranes, inability to bear fatigue, irregularity in the rise and fall of the internal temperature in spite of the appetite remaining comparatively good, absence of any localisation with the exception of irritation of the respiratory mucous membrane, and long period of convalescence, also belong, according to our experience, to pernicious anæmia. On the other hand, we have seen cases of pharynx-angina and contagious pleuro-pneumonia which closely resembled those of skalma as described by Dieckerhoff. It seems that enzoötic catarrh of the larynx and trachea have been described by some as skalma.

TUBERCULOSIS

General remarks on tuberculosis—Bovine tuberculosis—Tuberculosis of pigs—Equine tuberculosis—Canine tuberculosis—Tuberculosis of the cat—Tuberculosis of sheep—Tuberculosis of goats—Tuberculosis in wild mammalia—Tuberculosis of birds—Tuberculosis of parrots—Pseudo-tuberculosis.

A.—*General Remarks on Tuberculosis.*

Occurrence.—Tuberculosis (next to human beings) occurs most often in cattle, pigs, and fowls; and then in carnivora and horses. Its alleged infrequency among goats and sheep may be referred to a want of thorough investigation of the subject. It often occurs also in monkeys, lions, tigers, bears and other wild animals which are kept in confinement (the dromedary, giraffe, jackal, panther, jaguar, polar fox); likewise in rodents, and, it is said, in tortoises, frogs, lampreys, tench and carp. On the other hand, field-mice are immune. Also the native Japanese cattle are, in contradistinction to the foreign breeds introduced into that country and the crosses obtained therefrom, free from tuberculosis (Janson). Experiments made in the feeding of goldfish have had only a negative result.

According to von Behring, the susceptibility of the individual species of animals to tuberculosis is in the following order: the most susceptible are guinea-pigs; then rabbits, dogs, goats, cattle, horses, sheep and white mice.

The Tubercle Bacillus and Tubercle.—Tuberculosis is an infective disease that is caused by the bacillus tuberculosis, which was discovered by Koch in the year 1882. This bacillus is a slender rod of an average length of from 2 to 4 μ (about a third to a half of the diameter of a red blood corpuscle), 0·2 to 0·4 μ in thickness, and has rounded ends. It is easily stained with aniline dyes, and in that condition resists acids and alcohol. Artificial culture of tubercle-bacilli is difficult, and must be carried out indirectly from inoculated guinea-pigs. The bacilli thrive best and most rapidly on slightly acid nutritive media (brain agar and brain serum of natural acidity), on which they begin to grow after 24 hours, and form at the end of 8 days, dome-shaped or spherical colonies of 1–2 mm. in thickness ($\frac{1}{24}$ to $\frac{1}{12}$ of an inch); and at the end of 20 days, a cauliflower-like ridged and folded coating of 2–3 mm. ($\frac{1}{12}$–$\frac{1}{8}$ in.) in thickness, and of brownish-red or rosy-red colour.

Tuberculosis is anatomically characterised by the formation of nodular, cellular, non-vascular foci (tubercles). The development of tubercle according to recent investigations by Baumgarten is effected by the immigration of the bacilli into the tissues with consequent change in the nuclei (karyokinetic nuclear division), and proliferation of the fixed tissue cells, which lead to the formation of a great number of epithelioid cells that have one or two nuclei. These epithelioid cells form nodular masses, namely, tubercles (large-celled tubercle). The single cells are separated from each other by fine connective tissue fibres (the reticulum of the tubercle). Sometimes the epithelioid cells attain a great size, and then contain several nuclei (giant cells). Simultaneously with this cell proliferation, an emigration of leucocytes takes place around the tubercle. If the number exceeds that of the epithelioid cells, the tubercle is called small-celled or lymphoid.

The tubercle, when it has attained its full development, appears as a grey, translucent, hard nodule about the size of a millet grain (miliary* tubercle), which cannot be shelled out from the surrounding tissue, and which, on account of its non-vascularity, gradually undergoes retrograde metamorphoses, while its cells perish ; the tubercle becomes caseous or undergoes calcification. By the microscopical examination of a fresh typical tubercle we can, on making a section, easily distinguish three zones : 1. A central, round or angular, flat or spherical giant cell, with or without offshoots. It is granulated, and contains from 20 to 30 circumferentially-situated nuclei, and sometimes numerous tubercle bacilli. 2. A zone of large epithelioid cells which have round or oval nuclei, and which

* S. J. Gee and Percy Kidd (Quain's *Dictionary of Medicine*) remark : "Miliary tubercle may or may not be of the size of a millet seed. Tubercle the size of a millet seed may or may not be miliary. All that is now meant by miliary tubercle is a small nodule, roundish, seldom larger than a hemp-seed, almost colourless or greyish, consistence almost equal to that of cartilage, and either quite transparent or opalescent. Lastly, miliary nodules, may or may not be tubercular."

The miliary tubercle, which is the primary lesion produced by the bacillus tuberculosis, is non-vascular ; a fact which appears to be closely connected with the first degenerative change undergone by the grey granulation, namely, caseation. The subsequent retrogressive changes of calcification and fibrosis are methods of healing.

The use of the term "tubercle" (*tuberculum*, a small swelling) has, since the discovery of the tubercle bacillus, become more and more restricted to changes connected with tuberculosis. The expression "nodule" or "tumour" now, as a rule, replaces "tubercle" in the description of other diseases.—Tr.

contain 1 or 2 bacilli. 3. An outer zone with numerous small, round cells that have strong nuclei (granulation tissue) between which a fine reticular connective tissue lies. The giant cells and the epithelioid cells are sometimes absent.

Biology of the Tubercle Bacillus.—The well-marked power of resistance to various agents possessed by the virus of tuberculosis is due to the fact that the bacilli form resting spores. The tubercle bacilli perish very quickly outside the animal body in temperate climates; even sunlight of itself is able to kill them in periods varying from a few minutes to a few hours. Numerical increase or further development of the bacillus outside the animal or human body in the manner of a miasma is consequently impossible. Tuberculosis is therefore to be looked upon as a purely contagious disease. The tubercle bacilli are not ubiquitous (Cornet), and are comparatively resistant. They remain, for instance, virulent in ordinary water for 120 days (Cadéac); in the water of the Seine for 50 days according to Chantemesse, at 46° to 53° F.; and for 17 days at 59° to 64° F. Tuberculous sputum of man continued capable of infection, on one occasion, for 179 days; and on another, for 226 days. When in a dried condition it retains its infectiousness for some years (Struc). Dried tubercle masses from cattle continue infectious for about 100 days, when finely pulverised, and for 150 days when in pieces of the lung of the size of the fist. The same material resists decomposition for a period of from 76 to 150 days (Cadéac and Malet). In the gastric juice of the dog, tubercle bacilli retained their virulence from 4 to 6 hours, but were destroyed in from 18 to 24 hours. With respect to the influence of dilution on the action of tubercle bacilli, the investigations of Bollinger and Gebhardt have shown that the bacilli even after having been diluted to 1 to 400,000 produce tuberculosis when administered subcutaneously or intra-peritoneally. The fact of diluting the virus seems to proportionately decrease its power of infection only when it is given by the mouth, as in food. Dry sputum becomes sterilised in less than 15 minutes when it is exposed to steam at 212° F., and by dry heat in one hour (Schill and Fischer).

Concerning the height of temperature necessary to ensure the destruction of tubercle bacilli in milk, the results of various observers are conflicting. From the recent researches of Bang and Stribolt, a five-minutes' exposure to a temperature of 149° F. is destructive to tubercle bacilli. For large quantities, however, both recommend—in presence of formation of scum or mingling of large masses, or when the constituents are readily coagulable—a temperature of 185° F. Smith, Hesse, and Russel found that the bacilli were killed in from 15 to 20 minutes by a temperature of 140° F.—in a closed apparatus, indeed, they were destroyed by that temperature in 10 minutes. On the other hand, if milk were heated in open vessels, in which the formation of a surface-skin took place, the bacilli continued to live much longer, even up to an hour. According to Morgenroth, in order to destroy bacilli contained in it, the milk must be warmed to 158° F. for 30 minutes, or to 212 F.° for 3–5 minutes (*i.e.*, boiled). He also found that they were destroyed by heating to 131° F. for a period of 3 hours. Beck states that the bacilli are destroyed by boiling the milk for 3 minutes, but not less. Galtier testifies to the necessity of taking

the milk up to boiling-point; the result of heating it to 158–185° F. for 6 minutes being, according to him, uncertain. Barthel and Stenström state that a heating to 140° F. for from 5 to 20 minutes, to 158–167° F. for from 5 to 15 minutes, and to 176° F. for from 1 to 10 minutes, are all insufficient for the destruction of tubercle bacilli.

The effects of various disinfecting media on tubercle bacilli are as follows: sublimate disinfects dried sputa of tuberculous men after an exposure of from 20 to 24 hours to the influence of from 8 to 12 times the quantity of a solution of 1 : 1,000 to 5,000; so does also a 5 per cent. solution of potassium iodide, moist iodoform vapour, iodine vapour, mixing with iodoform, 1 per cent. soda-brine, and a 5 per cent. watery solution of creolin or carbol. Ineffectual, on the other hand, are: concentrated solution of common salt, 1 per cent. bromine water, aqueous solution of iodine (1 : 500), iodoform in oil or oil of turpentine (Schill and Fischer), alcohol, drying, freezing to 17.6° F., according to Galtier, and soap solution (Guinard).

Numerous observers have quite recently fully proved the existence in animals of radiating, fungiform growths of the tubercle bacilli (sprouting of the bacilli into threads with knotty ends). The assumption that the tubercle bacillus was, therefore, not a bifidate, but a filiform fungus, related to the actinomyces, has hitherto not been fully demonstrated; it is probably a question of age and involution.

General Remarks on the Pathogenesis of Tuberculosis.—As the immigration and the dissemination of the tubercle bacilli in the body are not uniform in all species of animals, and as cattle, pigs, and poultry, in particular, exhibit a special mode of invasion and distribution; we shall deal here only with the general principles of the pathogenesis of tuberculosis, and shall discuss special points and peculiarities more minutely when we refer to the respective species.

The tubercle bacilli enter the body most frequently along with the respired air* and through the alimentary canal. Consequently the lungs and the intestines are the chief ports of entrance for the virus. At first, tuberculosis always appears as a local affection which usually becomes developed at the place of entrance of the bacilli (lungs or intestines). However, it not unfrequently occurs that the organs through which the bacilli entered, especially the lungs and intestines, remain free from the disease, and that the tubercular process becomes

* Klein (*Micro-organisms and Disease*) states: "I have had guinea-pigs kept in their cages in the ventilating shaft of the Brompton Hospital [for consumptives], and have thereby produced general tuberculosis in the great majority of them: caseous tubercles in the lungs, lymph glands, spleen, liver, pelvic glands were the result; thus proving that the air of any place where tuberculous persons sojourn, contains the tubercle virus, and must therefore be considered as not free from danger."—Tr.

localised, first in the bronchial, or mesenteric glands, and from thence in the pleuræ or peritoneum, after the tubercle bacilli have passed through the respiratory or digestive mucous membranes without injuring them. The local affection consists in the formation of miliary tubercles, which, when in considerable numbers, may become combined together, so as to make a granulation tumour. These infectious new-growths can exist for a considerable time as localised foci of inflammation, or they may become calcareous, caseated, or undergo fibrinous degeneration. Or they may ulcerate, suppurate, or form cavities by disintegration. But the suppuration of the tubercle is not due to the tubercle bacilli, but to a simultaneous infection with the bacteria of suppuration (mixed infection). The disease may become cured by entire calcification of the tubercles, or by the formation of connective tissue completely separating them from the healthy parts.

From these local foci (localised tuberculosis), the tubercular process can, as is most frequently the case, spread in the following various ways, particularly in consequence of the breaking down of the tubercle. 1. By the *lymphatics.* Secondary tubercles spring up, first of all in the neighbourhood of the primary tubercle. After this, the lymph glands especially become infected ; in pulmonary tuberculosis, the bronchial glands and the mediastinal glands ; in intestinal tuberculosis, the mesenteric glands, spleen, and liver ; in tuberculosis of the upper air passages, tracheal glands, upper, middle and lower cervical glands ; etc. This affection of the lymph glands is always present in tuberculosis. The extension of the disease to the lymphatics very frequently goes on from the freshly inflamed lymph gland, to serous membranes, particularly those of the pleuræ and peritoneum. Furthermore, tuberculosis of the abdominal cavity, without the agency of the blood vessels, may spread to the thoracic cavity, by the bacilli passing through the finely fissured interstices of the diaphragm. 2. By *continuity*, especially by means of the *mucous membranes.* For instance, tuberculosis of the mucous membrane of the bronchi and larynx may, by the breaking into them of the neighbouring tubercular deposit, be at first set up, and later on tuberculosis of the intestine, from the swallowing of bronchial secretions which contain the bacilli. In a similar way, tuberculosis of the genito-urinary organs may become developed from the presence of the disease in the kidneys or genital glands. 3. Extension of tuberculosis by means of the *blood-vessels* may take place, when one of the large

veins, on becoming opened by erosion due to the breaking down of a tubercular focus (perhaps in the lungs), takes up part of the disintegrated mass and thus introduces it into the blood current; when a tubercular affection forms in the wall of a blood-vessel, as a consequence of the extension of the disease from a neighbouring focus; or when the bacteria are transmitted along with the lymph into the thoracic duct. In these cases, the bacilli may be carried by the blood into distant organs, and thus produce general tuberculosis (that is to say, it is distributed throughout the whole organism); they may be divided into two principal varieties, namely, chronic general tuberculosis, and acute, miliary tuberculosis. To *chronic general tuberculosis* we refer those cases in which, as a consequence of an injurious immigration of bacilli into the blood, only isolated tubercular foci form in various organs. These deposits at first are small; but subsequently they gradually enlarge by aggregation into nodules and tubercles. On the other hand, generalised tuberculosis is called acute miliary tuberculosis, when a sudden inundation of the blood with numerous bacteria takes place, or a rapid proliferation of the individual bacilli present occurs in consequence of an altered disposition of the affected animal. This *acute miliary tuberculosis* is characterised by the simultaneous invasion, in all organs, of innumerable minute (miliary) tubercles. The lungs and the liver are specially liable to tubercle on account of their comparative vascularity. Chronic general tuberculosis is not infrequently complicated with acute miliary tuberculosis. A mixed infection also takes place, especially in the later stage of tuberculosis, in such a way that other pathogenic bacteria, especially bacteria of suppuration (streptococci), are associated with the tubercle bacilli.

The somewhat rare cases of fœtal (placental) tuberculosis can be explained, partly by infection of the fœtus through the mother, the bacilli having penetrated the placental membranes, as has been experimentally demonstrated with animals (guinea-pigs and rabbits), and partly by a primary tubercular affection of the ovum at the time of coition. A germinal tuberculosis, namely, one inherited from the father, is extremely rare. The bacilli of this disease are very seldom found in the semen of tuberculous male guinea-pigs whose sexual organs are not the seat of tuberculosis. The number of tubercle bacilli in the semen of guinea-pigs which suffer from tuberculosis of the testicles, though somewhat greater, is still very small. When such animals were paired with healthy females, the

offspring was found to be thoroughly sound and free from tuberculosis (Gärtner, Seige). The widely spread assumption that male animals kept for stud purposes are an important factor in the dissemination of tuberculosis, by their transmitting the disease in the act of copulation, is therefore untenable. Hereditary predisposition to tuberculosis is a subject upon which scientific research has thrown no light.

Tuberculosis transmitted by Food.—The idea that tuberculosis is a specific infective disease is only of recent date; for the researches on this point spread over a period of rather less than thirty years. Prior to that time, tuberculosis was looked upon neither as an independent pathological unit, nor as an infective disease. It was only by numerous experiments in feeding and inoculation that the infective and uniform character of tuberculosis was clearly demonstrated. The first inoculation experiments were made by Villemin in 1865, from which time began the etiological investigation of this disease. Villemin transferred, partly subcutaneously, partly intra-tracheally, human tubercular matter to animals, and was the first to establish the fact that tuberculosis is a specific *infective disease, the cause of which is an inoculable agent.* He further asserted that tuberculosis of man was identical with the "grapes" or consumption of cattle. Klebs inoculated the serous cavities of the body and made the first experiments with food. By these means, he established the identity of tuberculosis of cattle with that of man, and was the first to lay stress on the danger of the milk of tuberculous cows being given to children. Chauveau (1868) also demonstrated, by numerous convincing experiments, the transmissibility of tuberculosis, by means of food, and declared that the consumption of the flesh of tuberculous animals was hazardous. The results of his researches were confirmed by Villemin, St. Cyr, and other French investigators.

Gerlach, along with Klebs, at the Hanover veterinary college in 1868, was the first in Germany to make exhaustive feeding experiments. After feeding with tubercles and with the milk of tuberculous cows, he succeeded in 7 experimental animals out of 8, in producing tuberculosis of the lymph glands, lungs, intestines, and liver. Gerlach had previously declared that the milk and flesh of tuberculous animals were infectious. He obtained, later on, similar results with 46 feeding experiments at the Berlin veterinary college. During the years

1870 to 1873, Günther and Harms undertook, in Hanover, 94 feeding experiments with animals of widely differing species and with various kinds of tubercular material, and obtained 24 positive results and 70 negative or doubtful ones. The majority of the 14 experiments made in 1870 and 1871 at the Dresden veterinary college gave positive results. The same may be said of the experiments of Zürn, Bollinger, and Roloff.

As these positive results were discounted by many negative ones; the theory of the transmissibility of tuberculosis by flesh and milk from animals to man, met with, at first, severe opposition and denial. Thus, in 1876 the German Veterinary Council rejected Gerlach's assumption. During the following year, positive results were produced by Blumberg, Lange, Orth, Toussaint, and Peuch, and by experiments at the Berlin veterinary college during the years 1876 to 1880, and at the Dresden veterinary college in 1878 and 1879. Besides, many cases of accidental transmission of tuberculosis through milk, sputum, and flesh, to men and animals were proved.

The most important year in the history of research in tuberculosis was 1882, in which Koch discovered the tubercle bacillus. From thenceforth the possibility of transmission was no longer doubtful. Thus, Baumgarten produced tuberculosis in every case by feeding with milk which had been mixed artificially with cultures of bacilli, and Haug invariably obtained positive results by experimental feeding with milk taken from tubercular udders of cows.

Various circumstances have contributed to render unsuccessful the numerous feeding experiments that have been made. In the first place, the material given as food frequently contained no tubercle bacilli, or extremely few. These bacteria, as is well known, are absent in caseous deposits and in calcareous bovine tubercles; in fact, bovine tubercles are, on the whole, poor in bacilli. The flesh also is seldom the seat of tuberculosis, and the milk, as a rule, only when the udder is affected. The virulence of experimental food was frequently weakened, or completely destroyed by putrefaction. The shortness of the time during which the bacilli remain in the stomach and intestines frequently prevented infection. In many cases, the experimental animals were more or less unsuitable. Finally, it appears that the dilution of the material containing the introduced bacilli by the other fodder, and especially by the drink and the digestive fluids, lessened the activity of the bacilli, which result

might also have been promoted by the direct influence of the digestive fluids. To clear up this point, Wesener made a series of feeding experiments by the direct introduction into the stomach and also into the intestines of a rabbit, of human tubercular sputum, respectively fresh, dry, putrescent, and after it had been subjected to the action of the various digestive juices. The investigations of Strauss and Würtz have also shown that the gastric juice weakens the virulence of the bacilli, and entirely destroys it in 6 hours. We may infer from these and other researches, that the natural gastric juice, if allowed to act for a sufficiently long time, weakens the vitality of the bacilli, and even kills them; but it certainly does not produce this effect on the spores, which may get into the intestine, and, without affecting the intestinal mucous membrane, may set up tuberculosis of the mesenteric glands (*tabes mesenterica*). In this respect, milk* which contains both bacilli and spores, is the most dangerous agent. Flesh is much less so; because, as a rule, it is free from bacilli, or contains them only in small numbers, and is usually consumed in a cooked state. Experimental inoculations which Bollinger made in guinea-pigs with the juice expressed from the flesh of tuberculous cattle, gave negative results. The experiments of Bang, Zschokke, Bollinger and others have shown that milk may contain bacilli or spores without the udder being affected, and may therefore act as an infective agent.

Wesener compiled reports, up to 1884, of 369 feeding experiments, the positive and negative results of which were about equal in number. We find from this compilation that—(1) 71 animals, among which guinea-pigs and swine proved most susceptible, were experimented upon with human tubercular matter. (2) 180 experiments were made with tubercular matter from cattle and other animals. More than three-fourths of the cases of inoculation were successful in calves, goats and sheep; three-fourths, in pigs; one-half, in rabbits; over one-third, in cats; and somewhat less than one-third, in dogs. (3) The flesh of tuberculous cattle was given on 32 occasions as food, with the result that pigs were found to be more susceptible than other animals, and that dogs were unaffected. Boiled flesh produced no effect. (4) The milk of tuberculous cows was given as food in 86 cases, out of which, three-fourths of the pigs and half of the goats became diseased. Boiling infected milk decreased its virulence. In the scale of comparative racial susceptibility, the herbivora (cattle, sheep, and goats) proved highest; then swine; and after them, guinea-pigs and rabbits. Carnivorous

* Klein suggests that the frequency of miliary tuberculosis in children is due to the ingestion of tuberculous milk. Sims Woodhead refers the high percentage of tabes mesenterica among children to the same cause.

animals (cats and dogs) were very little affected, and birds least of all with the exception of domestic fowl. The tubercular matter of animals was found to possess the greatest power of infection; then came the sputum of tuberculous men and the milk of tuberculous animals; and, lastly, tuberculous flesh.

Communicability of Tuberculosis of Domestic Animals to Man.—The question of the identity of tuberculosis of cattle with that of man, which, after the method advocated by R. Koch (*On the Etiology of Tuberculosis*, 1883), was usually answered in the affirmative, has entered on a new stage since the Congress of Tuberculosis held in London in 1901. In that year, Koch and Schütz, on the ground of the negative results obtained in experiments carried out at the Berlin Veterinary College, denied the identity of human tuberculosis with that of cattle, and therewith the communicability of tuberculosis of cattle to man. This new theory has received almost uniform contradiction. As the subsequent proofs of the Koch-Schütz experimental results, which have been undertaken in all directions, have not yet been concluded, a final decision on this question cannot at present be given. The assertion of the non-identity of human and bovine tuberculosis appears, however, to be already refuted by the positive results of numerous inoculation experiments.

The most important of the communications hitherto published on the subject are the following:

1. The *non-identity* of human and animal tuberculosis had been previously asserted by Pütz (1883) on the ground of negative results obtained in experiments on calves. The more recent researches of Koch and Schütz, undertaken to decide the question whether the bacillus of the human tubercle was distinct from that of tuberculosis of cattle ("grape disease"), were carried out in this way. The sputum of tuberculous men, and also pure cultures obtained from tuberculous human organs, were introduced into healthy experimental animals (tuberculin!) partly with the food and inspired air, and partly by subcutaneous, intravenous, intraperitoneal and intraocular injections. None of the numerous animals experimented on (calves, pigs, sheep) sickened after infection with the bacilli of human tuberculosis; while infection with the bacilli of bovine tuberculosis, according to the account of Koch and Schütz, always gave positive results. The final conclusion, especially with regard to cattle, was that *cattle were not susceptible to human tuberculosis*. As to the communicability of bovine tuberculosis to man,

Koçh has thus expressed himself: "Although the important question as to whether man is susceptible to bovine tuberculosis has not yet been decided, and the decision of the same will not be arrived at to-day or to-morrow, we are, nevertheless, already justified in saying at present that, even if a susceptibility actually exists, the infection of human beings thereby is of excessively rare occurrence. I would have it understood that the infection by milk, by butter made from it, and by the meat of tuberculous cattle can hardly be considered greater than that by hereditary transmission. I do not, therefore, consider it advisable that special measures should be taken against it."

Kossel, the Director of the Imperial Commission on Tuberculosis attached to the German Board of Health, has, on the ground of the researches there carried out, expressed himself in favour of Koch's assumption. He is, furthermore, of the opinion that "bovine tuberculosis does not play that part in the development of tuberculosis among men which has been generally attributed to it." According to the researches of Möller-Belzig, and of Theobald Smith, bovine tuberculosis is not communicable to cattle and goats. Frothingham and Dinwiddie found, besides positive inoculation results, that all calves were not susceptible to infection with human tubercle bacilli, while tuberculous material taken from men had little effect on cattle.

2. Nearly all the others who have carried out experiments, as well as the overwhelming majority of veterinary surgeons and medical men, have pronounced in favour of the *identity* of human and bovine tuberculosis. The London Congress of Tuberculosis (1901) came directly to the conclusion "that the question of distinction between human and bovine tuberculosis still remains undecided, and that no changes should be made in the existing regulations regarding the sale of the milk and flesh of tuberculous animals."

The following experts have up to the present individually expressed disapproval of Koch's view: in Germany, von Behring, Orth, Bollinger, von Baumgarten, Hueppe, Schottelius, Ostertag, Kitt, Johne, Albrecht, Karlinski, Prettner Weidmann, Klebs and Rievel, Schmaltz, Lassar, Max Wolff, Heller, Müller, Krause, Zahn, Sick, Priester and others; in England, Lister, McFadyean, Ravenel, Crookshank, Kingscote, Delepine, Faure, Still, Shennan, Grünbaum, and others; in France, Belgium, Holland, Denmark, Sweden, Russia, Switzerland, America, etc., Nocard, Arloing, Lignières, Chauveau, Montsarrat; de Jong,

Gratia, Thomassen; Bang, Jensen, Fibiger; Stensbröm, Svensson; Semmer, Zschokke, Salmon and others.

Von Behring has on the ground of the researches he carried out at Marburg during the past six years declared for the similarity of species of the respective bacilli of human and bovine tuberculosis. He specially points out that there exists in each a poisonous nucleus, the effects of which correspond to those of tuberculin, and expresses himself as follows: "As further arguments relative thereto I will enumerate: the absence of any distinction of impressive value in their morphology and culture; the identity of the anatomical and bacteriological conditions found in experiments on their communicability in guinea-pigs and rabbits; the transferability of human tubercle bacilli to cattle with positive consequences of infection; the possibility of obtaining, by appropriate transmission of the human tubercle bacillus, a high grade of virulence for cattle; the facts established by experiment that even bovine tubercle bacilli do not necessarily produce 'grape disease' (bovine tuberculosis) in cattle, and the conclusion which is more and more rapidly approaching, that the condition of tubercular swellings indicated by the term 'grape disease' is no more than an expression for a special form of the chronic course of the disease; the possibility of immunisation of cattle against the virus of bovine tuberculosis by means of human tubercle bacilli, and *vice versâ*; the possibility of artificial modification of not merely the virulence of a certain culture stock, but even its growth in cultures, as recognisable by the naked eye, till it arrives at the grade of the Arloing tubercle-cultures. In opposition to these arguments in favour of the similarity of the bacilli of bovine and of human tuberculosis, the reasons against that view seem to stand very far in the background as regards their strength."

Arloing has by injection of human tubercle bacilli into the veins of 23 specimens of large herbivorous animals (bulls, heifers, calves, sheep, goats, asses), made them experimentally tubercular, and has arrived, in opposition to Koch, at the following conclusions: 1. There is derived from the tubercle bacilli of man a pure culture, which can infect cattle, sheep and dogs with tubercle. 2. As the virulence of the tubercle bacillus varies, and as it possesses the peculiarity of adapting itself to many organisms, it is not surprising that the active agent of human tuberculosis displays a lower degree of infectiveness in many animals than does the bacillus of bovine tuberculosis. 3. When infection sometimes does not occur, it by no means follows that it depends on an absolutely different kind of tuberculosis. 4. We must accordingly adhere to the fact of the unity of human and bovine tuberculosis. 5. Koch and Schütz were not justified by their experiments in drawing so rigid a distinction between human and bovine tuberculosis. 6. It is accordingly necessary that the existing sanitary police regulations respecting meat and milk be allowed to continue.

Nocard is of opinion that tuberculous human beings more often infect cattle with the disease than it is transmitted by cattle to man. According to him, there are two degrees of virulence of human and animal tubercle bacilli; namely, a strong and a weak stock. These distinct grades elucidate the different results of experiments on cattle with human bacilli. The negative results of Koch prove nothing in presence of positive facts

(four positive inoculation experiments according to Chauveau). "These facts prove that, even although it be difficult to communicate human tuberculosis to cattle, yet it may sometimes be successfully done." "Even if we admit that cattle are practically not susceptible to human tuberculosis, have we a right to conclude therefrom that the reverse process is also unattainable ? This would be in opposition to all the fundamental laws of experimental methods. Above all things, it would not correspond to the facts." Doubtless healthy oxen have an extraordinary power of resistance to bovine tuberculosis, but they are not absolutely non-susceptible. If we inject cattle with a few drops of an emulsion of human bacilli, they die in about a month of tubercular meningitis.

De Jong has inoculated many experimental animals with tubercle bacilli derived from human beings, and among them 7 cattle, all of which became tuberculous. He has come, therefore, to the following conclusions: 1. The human tubercle bacillus can produce tuberculosis in cattle. 2. The human tubercle bacillus can also produce tuberculosis in other domestic animals (sheep, goat, dog) and in monkeys. 3. The tuberculosis produced in animals by the human bacillus is usually less grave than that developed from the bacillus of cattle. 4. The bacillus of cattle possesses a higher degree of virulence than that of man. 5. As this greater virulence is equally displayed by inoculation on cattle, sheep, goats, dogs and monkeys, we must conclude that the bacillus of cattle is also more virulent in the case of man. 6. As a source of infection, man plays a much less important part towards cattle than the latter do towards man. 7. On this account, bovine tuberculosis has a much greater importance in relation to human hygiene than has hitherto been attributed to it.

Thomassen has in 4 experiments produced tuberculosis twice (one calf and one heifer) by inoculation of human tubercle bacilli ; and has come to the conclusion that it was difficult, indeed, but not impossible, to produce generalised tuberculosis in cattle by pure cultures of bacilli which had been derived from man.

Schottelius, in the Hygienic Institute of the University of Freiburg, fed one cow and two calves with human tuberculous sputa. All three animals became tuberculous in the space of 4 months. In the case of the cow, there was tubercular enteritis established with great swelling of the mesenteric glands ; also tubercular caseation and calcification of the mediastinal and bronchial glands, as well as a tuberculous pneumonia with caseous masses and isolated miliary tubercles in the pleura ; caseous and calcified submaxillary glands, with scattered tubercular mesenteric glands.

Karlinski obtained positive results in 10 cases of inoculation of cattle with human tubercle bacilli. The communication succeeded 4 times by intraperitoneal inoculation ; twice by intrapleural ; once each by subcutaneous and intracheal inoculation, and by injection into the milk glands and testicles respectively (*15 other experiments gave negative results*).

Jensen and Fibiger succeeded 5 times in 10 experiments, in conveying human tuberculosis to cattle. According to Jensen, the communication of human tuberculosis to cattle is very difficult, but not impossible. All tubercle bacilli belong to one and the same species, which produces varieties more or less stable that have displayed themselves in course of time. The researches of Koch are, according to Jensen, in no way capable of influencing the previous conception of the relationship between bovine and human tuberculosis. It is firmly established that human beings may

be infected with tuberculosis in dissection of tubercular cattle (through skin-wounds). Bang has also conveyed human tuberculosis twice to cattle.

Klebs and Rievel have successfully inoculated a calf with human tubercle bacilli ; so has Max Wolff ; and Prettner obtained positive results with 3 calves.

Svensson and Stenström have conveyed the infection to 3 experimental calves ; they both hold that cattle are easily infected with human tuberculosis ; but human tubercle bacilli are relatively less virulent to cattle.

McFadyean does not deny that according to the results of inoculation experiments, the tubercle bacilli of cattle are more virulent than those of man. The difference in the virulence does not, however, prove a difference of species. Experiences in English hospitals for children (frequency of tuberculosis in children) point to bovine tuberculosis as a source of infection. The experiments recently carried out on cattle at the London Veterinary College prove the possibility of the infection of cattle by bacilli which have been derived from a human source. The danger of cattle becoming infected through the natural channels by tuberculous human beings is, however, but slight (Kingscote).

Delepine inoculated 2 calves with positive results ; according to him, it is beyond doubt that animals may become affected with tuberculosis from human tubercle bacilli.

Hueppe regards Koch's conclusions as not proven, and refers to the inoculation experiments carried out by Bollinger, who produced pure "grape disease" (bovine tuberculosis) in calves with tubercular products obtained from human beings. The tubercle bacillus so adapts itself to the organisms of its various hosts that, for example, when it has become a human bacillus it is not at all so virulent to another species of host, *e.g.*, to cattle. This variety in the feeding-grounds of the parasites does not disprove the identity of the various species of bacilli found in mammals, nor the dangers associated therewith.

Von Baumgarten has had negative inoculation results, and so far agrees with Koch that he sees in the tuberculosis of cattle no very great danger. He would, however, regret if the firm belief in the unity of human and bovine tuberculosis which had been established were, on the ground of these negative results, abandoned in the absence of further proof ; and he recommends that the precautionary measures hitherto established for the protection of human beings from bovine tuberculosis be still rigorously maintained.

B.—TUBERCULOSIS OF INDIVIDUAL DOMESTIC ANIMALS.

1.—(*Bovine Tuberculosis.*)

History of Tuberculosis in Cattle.—Tuberculosis of cattle is one of the oldest known diseases of our domestic animals. The Mosaic laws (Leviticus xxii. 22) contain rules that the flesh of animals which suffer from "wen or scurvy" should not be used as food. The Talmud, especially the Mischnal (third century), and the Gemara (fifth century), contain numerous enactments against the eating of such flesh. The

"kandi" and "timari" in these books probably mean tubercle. It is evident that in the eleventh and twelfth centuries, tuberculosis was fairly well known to the Arabian Rabbi Isaak Alfasi, the Hebrew physician, Maimonides, and the French physician Raschi (1105). The book, *Schulchan Aruch*, which is a Jewish collection of rules on this subject, has been handed down to us from the sixteenth century.

The Franks had in Germany, in the ninth century, ecclesiastical laws against the eating of the flesh of cattle and pigs which were affected with tuberculosis of the serous membranes (*kadrerie*). In the year 1370 it was forbidden in Munich to have on sale the flesh of tuberculous animals. Similar laws were passed in 1343 in Würtzburg; in 1394, in Passau; in 1401, in Landshut; in 1558, in Würtemberg; in 1582, in the Palatinate; etc. It is reported that 12 students who had eaten the flesh of tuberculous animals, died in Leipzic in 1677. In 1702 Florinus gave a description of the symptoms of the disease during life. Even at that time the name of "French disease" was in common use. The name arose from the fact that tuberculosis was considered to be connected with human syphilis, which was regarded as its cause (bestiality). The term *Franzosenkrankheit* is said to have been first used by Helmont. In consequence of this theory of origin, all tuberculous cattle had to be destroyed. Thus, for instance, the yearly loss arising from the slaughter of affected cattle in the Prussian countries amounted, according to Graumann (1784), to about £900. Severe penalties were enacted against the violation of the sanitary laws.

In 1783, the Berlin Board of Health first published inspection of meat regulations, in which it described the characteristics of "French disease;" rejected the theory of its connection with syphilis, and declared the flesh to be fit for human food. In the same year, Kersting of Hanover expressed a similar view in a report to the government of Mecklenburg-Strelitz. Franck of Baden was of opinion that the private consumption of the flesh of cattle which were only slightly affected with tuberculosis might be permitted. Graumann declared in 1784, in a government order of Mecklenburg-Schwerin, that such meat might be eaten. In consequence of this, all the orders which had been made against the consumption of the flesh of tuberculous cattle were cancelled throughout Prussia in 1785; and throughout Austria in 1788. Tscheulin, in 1816, laid down with regard to the inspection of meat, three degrees of bovine tuberculosis:

(1) in which only the tubercles were to be removed ; (2) in which the diseased parts were to be destroyed and the meat to be sold at a cheap rate ; and (3) in which the flesh was declared to be entirely unfit for food. A similar procedure, as regards meat inspection, was carried out, up to a very recent date, in Southern Germany, Switzerland, Austria, France, Belgium, Spain, Italy, and other countries.

Of the many terms employed in earlier times to denote bovine tuberculosis, the following may be mentioned : lung-consumption, lung-disease, grape-disease, millet disease, duck-weed disease, mallenders, beardiness, scratchings, flotchings (= measles), cattle shame, gland disease (= strangles), pocky disease, French disease, syphilitic disease, venereal disease, disease of lechery, bull's disease, menstrual cavalcade, lymphosar comatosis.

Phthisis and bovine tuberculosis were for a long time considered to be two different diseases. Thus Virchow, Schüppel, and others declared that the tubercles in cattle were lymphosarcomata. Leisering regarded them as sarcomata. On the other hand, Spinola and Haubner maintained that bovine tuberculosis and human tuberculosis were identical. This identity has, however, only during the last decade, been positively proved by the feeding experiments of Gerlach and the bacteriological results of Koch.

Etiology.—The penetration of the tubercle bacillus into the tissues of the body is undoubtedly the sole cause of the disease, although the mode of infection may vary. Usually in case of adult cattle the infection is transmitted by cohabitation or by inspiration of tubercle bacilli. The occurrence of lung tuberculosis and tuberculosis of the bronchial glands in older cattle bears testimony to this mode of infection. Thus many cases are known in which a new-comer infected in turn all the other residents of the cow-shed. In such case the infection takes place from coughed-up tubercular masses (inhalation tuberculosis) which are either dried and mixed in the form of dust with the inspired air (dust-infection), or by the force exerted in coughing, are broken up into fine particles which are retained floating (undulating) for some time in the moist atmosphere of the shed in the form of minute bubbles (drop-infection). Bacteriological researches by Ravenel have shown that tuberculous cows expel in coughing small virulent particles in which the presence of tubercle bacilli can usually be demonstrated. On

the other hand, the researches of Cornet, Cadéac, Malet, Celli and Guarineri show that we must not assume that the tubercle bacilli are exhaled into the air by the affected animals and are then inhaled in a pure condition by their neighbours. In a smaller number of cases the transmission of the bacilli might take place *per os* by the animals licking each other and by the consumption of sputum and bacilli which have been deposited from the air in the form of dust on the fodder (Bang, Cadéac, Bournay). This theory receives support from the fact that in a cow-shed which has a common fodder trough * the tuberculous animals which stand above the others infect them, and that the spread of the disease can be stopped by simply changing the position of the cattle.

In young cattle, and especially in calves, the infection takes place most frequently through the digestive apparatus (food-tuberculosis). The proof of this fact is furnished by the predominant disease of the digestive organs and of their lymphatic glands. The disease is very often transmitted to calves by the milk of tuberculous cows, especially when the udder is affected. The milk of tuberculous cows may, as we have already said, contain bacilli and spores without the udder being diseased. Investigations of Heim, Leser, Bang, Roth and others have also shown that dairy produce (whey, cheese, and butter, for instance) from tuberculous cows may transmit the disease, even after a lapse of from one to four weeks. Bang has proved that although the centrifugal process deprives tubercular milk of a large amount of its poison, by hurling the greater number of the bacilli into the sediment, it does not afford absolute protection from infection; because skimmed milk and cream prepared by centrifugal methods have, on inoculation, been found capable of setting up tuberculosis.

The existence of congenital (fœtal) tuberculosis has been amply proved by a large number of observations. Here we must distinguish between a placental infection, namely, the infection of the embryo through the diseased mother, and a conceptional or germinative infection, namely, the infection of the ovum, through the semen, during coition. The possibility of placental infection has been proved by frequent veterinary observations. Its occurrence depends on a previously diseased condition of the placenta as Sanchez has shown experimentally with pregnant rabbits, which, by inoculation, contracted the disease without the fœtus becoming affected, as long as the placenta remained

* The trough here alluded to has a downward slope from one end.

intact. The rarer germinal affection has been shown to be possible by the experiments of Marfucci, who inoculated hens' eggs with cultures of the bacilli and obtained well-marked tuberculosis in the chickens. The cases of tuberculous infection during the first months of fœtal life are, as a rule, not rare; though they generally lead to early abortion; although the disease has on several occasions been found in newly-born calves. Infection through coition from the bull to the cow, and *vice versâ* is less frequent; though the possibility of its occurrence has been proved by many practical observations.

The entrance of the tubercle bacilli occasionally takes place through the skin and the subcutaneous tissue (primary tuberculosis of the skin and subcutaneous tissue, also of the neighbouring joints, tendon sheaths, and mucous sacs). It is likewise possible that the udder may become primarily infected by the tubercle bacilli penetrating through the canal of the teat (Bang).

The bacilli may penetrate into the body through intact mucous membranes. This entrance is facilitated by certain predisposing influences, which were formerly considered to be the real cause of tuberculosis, such as feeding on innutritious and watery food, and prolonged residence in badly ventilated and crowded sheds. The fact that depastured cattle, especially on mountains, become much less frequently affected with tuberculosis than stall-fed cattle, is the best proof that the vitality of the respiratory organs becomes weakened in cattle which are kept in sheds, and which consequently become predisposed to the penetration and deposition of tubercle bacilli in their bodies. The penetration of the bacilli is greatly facilitated by the presence of catarrhal affections of the respiratory mucous membrane, which are caused or favoured by the state of the air in the shed, especially when there is an accumulation of secretion in the bronchi. The disease caused by arsenical fumes is, according to Johne, a true chronic inhalation-tuberculosis, arising from the inhalation of these fumes first of all setting up an inflammatory affection of the mucous membrane, which facilitates the invasion of the bacilli. The predisposition to tuberculosis is also increased by an abundant production of milk and numerous births, both of which weaken the system and impair its power to resist the penetration of the bacilli. For similar reasons, the disease frequently becomes much worse directly after a birth, or gets well marked when puberty is attained. Continuous in-breeding and attempts at establishing

pedigree stock are predisposing factors which account for the susceptibility to tuberculosis of animals bred from closely related parents. An inherited predisposition seems not uncommonly to occur by reason of the constitutional debility of the mother being transmitted to the offspring.

Occurrence.—Tuberculosis is by far the most widely-spread bovine disease. It is found in nearly all countries and regions of the world, and most frequently in the neighbourhood of cities. In the polar regions, the North of Sweden and Norway, North Africa (Texier and Cochez), the steppes of Russia (Spinola), and in some isolated and but little frequented islands, as for instance, Iceland (Krabbe) and Sicily (Chicoli), the disease occurs very seldom or not at all. As we have already said, it does not appear among Japanese cattle (Janson) or wild cattle (Veith). The fact is accepted in Germany that in secluded districts and parishes in which close attention is paid to maintaining the purity of the local breeds, tuberculosis is rarer than in places where there is much traffic in cattle and where foreign cattle are imported. Bang made similar observations respecting certain Danish islands; and Krajewski, as regards the grey cattle of the South Russian steppes.

The *percentage of cattle suffering from tuberculosis* varies much in different districts. In Germany in 1900 the percentage was 25; but according to the statistics collected in the year 1888-1889 by the Imperial Board of Health only from 2 to 8 per cent. of all the cattle were found to be affected with tuberculosis. These figures are certainly far too low; for the positive results with tuberculin inoculations have not infrequently been as high as 50 per cent. The percentages in the different states were in 1888: Prussia, 5; Saxony, 8; Bavaria about 3; and Baden and Hesse, 2. In Prussia the 5 per cent. of the total number of cattle corresponds to from 6 to 7 per cent. of all the cows. The different provinces show the following percentages: Pomerania, 16; Magdeburg, 12; Lower Silesia, 11; Schleswig-Holstein, Upper Silesia and Posen, 7 to 10; Hanover, Brandenburg, Bromberg, Breslau, Erfurt, and Merseburg, 3½ to 5; West and East Prussia, 3; Westphalia, 2; and the Rhine Province, 1 to 2½. Tuberculosis was accordingly most prevalent in Pomerania and least so in the Rhine Province.

Since that date tuberculosis has generally increased among cattle. Eight per cent. of all the slaughtered cattle were

found to be tuberculous in Prussia in the year 1893–1894 (64,000 tuberculous among 777,000 slaughtered); in the districts of Magdeburg and Stralsund, 16; Coblentz, 12; Berlin and Lüneburg, 11; Marienwerder, Cöslin, Liegnitz, and Oppeln, 10; Merseburg, Hildesheim, Wiesbaden, and Aurich, 9; and Danzig, Frankfurt, and Stettin, 8. In late years tuberculosis has increased in Prussia to an extraordinary degree. The percentages of slaughtered cattle which were found to be tuberculous reached an average of 15 in the years 1895 to 1900 (13 per cent. in 1895, 14 per cent. in 1896, 15 per cent. in 1897, 14–15 per cent. in 1898–9, 17 per cent. in 1900, and 15 per cent. in 1901). In the abattoirs of Saxony in 1889, the percentage of tuberculosis in all the cattle slaughtered varied from 1 to 16; *in the year* 1888, *from* ½ *to* 22½, *and in* 1892, 1893, and 1894, from 18 to 21½; in the years 1895–1900 it reached from 26 to 30 per cent. In 1897, 30 per cent. of the cattle slaughtered in Dresden were found to be tuberculous. Of the cattle slaughtered from 1888 to 1900 in Leipsic, an average of 27 per cent. (11 to 35 per cent.) were affected with tuberculosis. In Bavaria, the proportion of slaughtered cattle found to be tuberculous during the years 1895 to 1900 was from 5 to 9 per cent. The number of animals found to be tuberculous in the abattoirs in Munich and Augsburg amounted to from 2 to 11 per cent. It was found in England during the year 1892, when cattle were being slaughtered on account of contagious pleuro-pneumonia, that from 20 to 30 per cent. were tuberculous. The percentage in London was from 25 to 40; and in Edinburgh, Yorkshire and Durham, 19 to 23. The tuberculin inoculations in England in the years 1897–1900 gave an average of 25 per cent. of cattle which reacted. The percentage in the slaughter-houses at Buda-Pesth reached 13 in the year 1900. In Russia a large percentage of the foreign milch-cows which were kept in stalls were tuberculous. In Kiel, from 20 to 40 per cent. of the Danish cattle slaughtered (cattle quarantine) during the years 1895–1900 were found to be affected with tuberculosis.

Lowland *breeds*, such as Dutch cattle, are much more frequently affected than the mountain breeds, which possess stronger constitutions. Cattle kept in sheds, as we have already remarked, are far more susceptible than those on grass. With regard to sex, we find that cows and heifers are more liable to tuberculosis than bulls and oxen. We learn from the foregoing statistics that out of every 223 animals, 170 cows, but only a? *oxen,* 14 *bulls, and* 1 *calf suffered from tuberculosis. It is*

likely that this disease is most prevalent amongst the milch cows of dairies, distilleries, breweries, sugar refineries, etc. Old animals are much more frequently tuberculous than young ones. Of the aged milch-cows, an average of 50–75 per cent. are tuberculous. According to the before-mentioned statistics, the cases of bovine tuberculosis, as regards age, during 1888 and 1889 in the German Empire, were as follows: 43 per cent. were 6 years of age; 33 per cent. from 3 to 6 years; 11 per cent. from 1 to 3 years; 1 per cent. under 1 year old; and 0.4 per cent. under 6 weeks. Among a million calves which were killed in Munich from 1878 to 1882, only 5 were found to be suffering from congenital tuberculosis. In the years 1896–1899, the tuberculous proportions of the slaughtered calves in Prussia, 0.15 to 0.1 per cent., was in the previous four years 0.08, 0.05, 0.04 and 0.02 per cent. respectively. In Bavaria, the number of tuberculous calves in the years 1895-1899 reached a proportion of 0.02 to 0.05 per cent : in Saxony, 0.15 to 0.25 per cent. In Baden, of 1,500,000 calves slaughtered in the years 1888–1898, 0.35 per cent. were tuberculous. Among about 80,000 calves that were slaughtered in Berlin in 1885–1886, only 7, and amongst about 90,000 killed in 1886–1887, only 6 were tuberculous; in the years 1895–1897, the percentage at Berlin was 0.1 to 0.6. Only 9 cases of tuberculosis were met with in 230,000 calves which were killed in Augsburg from 1873 to 1886. On the other hand, at Kiel, among 4,000 healthy calves, 26 (*i.e.*, just over ½ per cent.) were found to be suffering from hereditary tuberculosis. The percentage of cases of tuberculosis in newly-born calves is reduced by the fact that tuberculosis is an exciting cause of early abortion. Buffaloes in the wild state would appear to be seldom or never affected with tuberculosis. Among 5,000 slaughtered at Prague not a single case of tuberculosis was found to exist (Prettner). On the other hand, the disease also occurred in buffaloes, when kept in sheds; and especially when placed in stalls which had been occupied by cows (de Bendictis, Kanzelmacher).

Anatomical Conditions.—The anatomical changes in bovine tuberculosis are mostly found in the lungs (phthisis) and in the serous membranes of the large cavities of the body ("grape disease").* In about half of all the cases, the lungs and the

* In Germany, the expression *Perlsucht* (pearl-disease) is used to signify tuberculosis of the pleuræ and peritoneum, and corresponds, more or less, to the English term, "grape disease." We may translate *Perlen* by "grapes."

serous membranes became simultaneously affected; in about a third, the lungs alone; and in about one-fifth, the serous membranes only. When the lungs and serous membranes are attacked their respective lymph glands always become affected at the same time. In constitutional general tuberculosis, all the other organs of the body may show tubercular changes, which, however, may be restricted even to one organ (compare the figures on page 355, Vol. II.).

1. In the *lungs* we find, first of all, circumscribed caseous pneumonic foci of various sizes, which develop from a catarrhal pneumonia, with atelectasis (imperfect expansion) of the alveoli and bronchioles, emigration of leucocytes, and accumulation of large epithelioid cells in the interior of the alveoli (desquamative pneumonia), and subsequent caseation or suppuration of the affected parts of the lungs. This gives rise to the formation of caseous foci and cavities with yellowish, caseous, crumbling, and greasy or purulent contents. These changes are accompanied by chronic indurating inflammatory processes in the interstitial tissue of the lungs. These processes appear chiefly in the neighbourhood of the caseous foci and miliary tubercles; and manifest themselves by an interstitial new growth of connective tissue, and carnification and shrinking of the lung tissue, which sometimes shows an almost cartilaginous or flesh-like thickening and even complete calcification, and consequently offers a certain amount of resistance when it is being cut through.

Another kind of tuberculosis of the lungs manifests itself, in the presence of the miliary tubercles, as nodules, varying in size from a millet seed to a pin's head. They are pale-yellow, of moderately firm consistence, and are only slightly translucent. They are usually spread in great numbers, either over the surface of the lungs, or prominently on the surface of the section; and at first are surrounded by thoroughly healthy lung tissue. Later on, these nodules become caseated and calcareous in their centre. Large tubercular nodules are frequently formed by the accumulation of several of these miliary tubercles, which, in old standing cases, may undergo caseation or calcification in their interior.

The bronchial mucous membrane is often in a state of chronic catarrh in which greater or less bronchiectasis (dilatation of the bronchi) is not rare. Also, tubercular ulcers and miliary tubercles may occur in the bronchial mucous membrane and in the laryngeal mucous membrane; the

ulcers being distinguished by thickened, turned-up edges, and the miliary tubercles by the fact of their being arranged in rows. The tubercles spread usually from the bronchial mucous membrane to the peribronchial connective tissue (bronchitis and peribronchitis nodosa) and also to the lung tissue. Sometimes tuberculous new growths are found in the larynx, also ulcers, nodular hyperplasia of the mucous membrane, with infiltration and abscess-formation in the sub-mucous tissue, the parachondrium and the laryngeal muscles.

The bronchial glands are always swollen, enlarged and infiltrated with miliary tubercles. Later on they become caseous and calcareous. On the pleuræ there may become developed a circumscribed, chronic, adhesive pleuritis, which is followed by adhesion of the lungs to the thoracic wall. Sometimes the lungs show at the same time other specific changes, such as those caused by echinococci, pleuro-pneumonia, etc.

2. Tuberculosis of the *pleuræ* and *peritoneum* ("grape disease") begins with the formation of very minute, light grey, translucent nodules (" grapes ") which are at first smaller than even a grain of coarse sand, and give rise to the granulated condition of the surface of the pleuræ and peritoneum. An abundant new-growth of connective tissue forms round these tubercles (" grapes ") with greatly increased vascularity of the serosa, so that the tubercles are, as it were, imbedded in a frame of connective tissue.

On account of the confluence of several of such tubercles and the simultaneous proliferation of the connective tissue stratum, nodules, varying in size from a lentil up to a pea, now form partly on the visceral and partly on the parietal membrane, and may finally become as large as a hen's egg or as a man's fist. At first they are of a soft, juicy, gelatinous consistency, and of an orange colour, and, on a section being made, often show a dark-red centre. Later on they become harder and firmer, assume a fibrous or connective tissue-like consistency, and acquire an uneven nodular surface, so that they grate on being cut through. Or they undergo caseation or calcification in their centre, which then consists of a brittle, mortar-like, grey-yellow substance. In this more advanced state, the colour of the tubercles is light grey, ash-grey, or bluish white. These tubercles (" grapes ") assume very characteristic forms. By large numbers of them being variously grouped together,

they may present a villous, wart-like form, or racemose appearance; or they may take the shape of a cauliflower or mulberry. Sometimes they have a broad base, at other times they are pedunculated or shaped like a pendulum or polypus. They may cover the entire surface of the serous membrane, and may become enormously increased in number. These tubercular new-growths frequently attain a weight of from 66 to 88 lbs. and even more. The individual nodules have, at this stage, either grown firmly together, or have become united by bands or threads of connective tissue.

The anterior and posterior *mediastinal glands* become enlarged in the same way as the bronchial glands. They become infiltrated with miliary tubercles, indurated or caseous, and changed into tumours of greater or less size, even up to half a yard long. The enlarged mediastinal glands frequently enclose and compress the œsophagus.

3. Besides the bronchial and mediastinal glands, a considerable number of the other *lymph glands* of the body usually undergo tubercular changes in the manner described. On the head, the glands usually affected are those of the larynx and the lymph glands of the parotid region; on the neck, the upper (retropharyngeal), middle, and lower cervical glands; in the anterior extremities, the glands of the shoulder [glandulæ cervicales superficiales hom.], prepectoral glands [glandulæ axillares], and brachial glands; in the posterior limbs, the inguinal glands, iliac glands [glandulæ iliacæ externæ hom.], and popliteal glands; on the udder, the pudic glands (supramammary glands); on the croup, the external pelvic glands; in the thoracic cavity, the intercostal and sternal lymph glands, and the bronchial and mediastinal glands; in the abdominal cavity, the glands of the mesentery, lumbar region, liver, spleen, kidneys, etc. The affected glands may sometimes attain a very large size.

4. In the *abdominal cavity*, besides the implication of the peritoneum (parietal membrane, omentum, and mesentery) and lymph glands, we may have tubercular changes in various other organs, such as the liver and spleen, which will then contain small and large tubercles, and caseous foci. Tuberculosis of the spleen commences usually with a uniform swelling of the Malpighian corpuscles (follicular hyperplasia). The tubercles themselves develop in the lymph follicles and in the perivascular connective tissue. The kidneys not unfrequently suffer from tuberculosis, which causes them to become enlarged and often

intermingled with numerous yellowish-white tubercles varying in size from a millet seed up to a pea. These tubercles undergo in the centre caseous disintegration and become surrounded by a capsule of connective tissue. According to Schütz, their development takes place in the interstitial tissue, during which process the normal component part of the kidneys becomes destroyed. At the same time, parenchymatous or hæmorrhagic nephritis may be present. Sometimes the kidneys appear externally sound, while their interior is studded with tubercles. On the other hand, tuberculosis of the capsule of the kidney is occasionally observed. The ovaries frequently become enlarged into tumours the size of a man's head. Ulcers are found in the mucous membrane of the affected *uterus*, which may, as a consequence of the disease, assume large dimensions; the nodules being situated either in the subserous intermuscular or submucous connective tissue and may break through into the uterine cavity. Sometimes we find a tubercular degeneration of the placenta. In the uterus there is generally a turbid, ichorous fluid which is heavily charged with bacilli; on the serous surface there are sometimes tufty outgrowths. The oviducts are frequently closed up by tubercular growths, forming serpentine cords of the thickness of the finger. Tubercular processes also take place in the vagina and in the vulva. In the testicles are tubercular nodules, smaller and larger nodules, with caseous foci, to be found, which may be associated with tuberculosis of the tunica vaginalis and hydrocele. The epididymis, seminiferous tubes and the prostate, as well as the vas deferens, may be also affected.

The mucous membrane of the *intestine*, especially of the large intestine, sometimes displays tuberculous ulcers and nodules (Sanfelice); these are rarely found in the rennet-bag (Kitt, Prietsch, Rieck), or the manyplies (Johne). Nodular masses also develop under the mucous membrane and the serous coat of the stomach and intestine. Johne, Frothingham and others have described in cattle a peculiar infiltrated form of tuberculosis of the intestine without ulcerous formation, which is difficult to recognise macroscopically. Tubercular changes sometimes occur in the tonsils (Winter).

5. Tuberculosis of the *udder*, in cases of chronic general tuberculosis, appears as a diffuse, firm enlargement, in which case, according to Bang, the gland lobules, on *post-mortem* examination, appear swollen, uniformly grey coloured, and studded with small yellow points and striations, and small

hæmorrhages. The larger milk ducts contain yellowish, caseous masses full of bacilli. Later on, we find in the udder a considerable increase of connective tissue (chronic, localised, interstitial mastitis) and firm tubercles, which are of various sizes and which become caseated or calcareous. Consequently the udder feels nodular, firm (sometimes almost as hard as a stone), and may attain an unusually large size, weighing up to 44 lbs. or more. In the wall of the milk reservoirs and larger milk canals, numerous miliary tubercles are present. Enormous deposits of tubercle bacilli may be found everywhere in the udder. All the lymph glands lying above the posterior region of the udder are enlarged in every case, and may become indurated or caseous.

6. Tubercular changes are met with in the *brain* and *spinal cord* more frequently than is usually believed. Semmer, for instance, observed 4 cases of cerebral tuberculosis among 40 tuberculous cows. Tubercles, mostly of different ages, and varying in colour from grey to yellow, and in size from a millet seed up to that of a hen's egg, may form in the pia mater, arachnoid, brain substance, and inner wall of the ventricles. These tubercles are most commonly found in the pia mater at the base of the brain, where they may remain for a considerable time without producing any further inflammatory changes; although, not unfrequently, they give rise to inflammation of the membranes of the brain (leptomeningitis basilaris tuberculosa). The membranes of the brain may become adherent to each other and hydrocephalus internus may ensue. Sometimes the tubercles unite so as to form large masses. Similar changes manifest themselves in the spinal cord, in which the pia and dura mater are studded with numerous small tubercles, and frequently become adherent to each other. Even superficial ulcers, caused by pressure, have been observed on the wall of the spinal canal (Johne).

We sometimes meet with tubercular changes in the *eyes* (tuberculosis of the iris, choroid, and retina; tubercular *phthisis bulbi, i.e.*, conversion of the bulb into a caseated or granulating mass); *muscular system* (miliary and large tubercles of the muscles of the skeleton, especially in the muscles of the croup, abdomen, chest, and heart, with adhesions of the heart to the pericardium); and in the *bones* (petrous portion of the temporal bone, frontal bone, occipital bone, cervical vertebræ, especially the first and second, lumbar vertebræ, dorsal vertebræ, ribs, sternum, and the large hollow bones); and in the cartilages

(nasal septum, cartilages of the ear). Generally, tuberculosis of the bone starts from the medulla (osteomyelitis granulosa), and runs its course by the formation of reddish-grey granulation foci and lucunar breaking down of the bone tissue (caries), with central caseation and formation of cavities. According to Hess and Guillebeau, tubercular processes also frequently occur in the joints (vascular, fungal, granular and caseous synovitis, especially in the knee and carpal joints); tendon sheath (of the extensor carpi radialis and extensor pedis longus muscles), and mucous sacs (anterior aspect of the carpal joint). They are more rarely found in the larger vessels; *e.g.*, in the wall of the aorta, in the skin, and in the subcutaneous tissue (nodules, abscesses, ulcers, lymphangitis).

The state of nutrition, especially in cattle suffering from tuberculosis of the serous membranes, is often good, notwithstanding the existence of extensive tubercular changes. There is always, however, considerable emaciation and anæmia, and finally hydræmia, in cases of advanced tuberculosis of the lungs.

Statistics.—The researches made from 1888 to 1889 in the German Empire, respecting the seat of tubercular changes, show the following results:—One organ only was affected in from 50 to 60 per cent. of the cases; one cavity of the body, in 13 to 17; several cavities, in 15 to 20; and the flesh, in about ½ per cent. General tuberculosis was present in 10 per cent. The following table gives the comparative frequency of the disease in different organs:—

1.	Lungs	75 per cent.
2.	Visceral pleuræ	55 „
3.	Peritoneum	48 „
4.	Costal pleuræ	47 „
5.	Bronchial and mediastinal glands	29 „
6.	Liver	28 „
7.	Spleen	19 „
8.	Uterus	10 „
9.	Lumbar glands	5 „
10.	Pharyngeal glands	4 „
11.	Trachea	3 „
12.	Udder	1 „
13.	Intestines	1 „
14.	Ovaries	1 „
15.	Lymph glands of the liver	1 „
16.	Lymph glands of the thoracic and abdominal cavities	0·9 „
17.	Heart	0·9 „
18.	Kidneys	0·7 „
19.	Bones	0·4 „
20.	Internal viscera (including the brain)	0·3 „

21. All the organs of the thoracic and abdominal cavities	0·2 per cent.
22. Submaxillary glands	0·2 ,,
23. Diaphragm	0·2 ,,
24. Stomach	0·16 ,,
25. Larynx	0·13 ,,
26. Muscles	0·1 ,,
27. Iliac and inguinal glands	0·06 ,,
28. Brain	0·04 ,,
29. Spinal cord	0·03 ,,
30. Tongue	0·01 ,,
31. Thymus gland	0·01 ,,
32. Vagina	0·01 ,,
33. Testicles	0·01 ,,

According to Bang, 0·5 per thousand of all cows in Denmark suffer from tuberculosis of the udder; to Ostertag, 0.5–1 per cent; and to Schütz, 0.3–1 per cent.

Pathogenesis of Tuberculous changes in the Organs.—R. Ostertag has utilised the records of the Berlin abattoir for procuring exact information on the pathogenesis of tubercular changes in the individual organs of cattle, as compared to those of pigs, and for obtaining a definition of local and general tuberculosis. According to these researches, the primary affections in cattle take place, most frequently, from the respiratory apparatus and from the uterus. Tuberculosis of the pleuræ and peritoneum may, however, often occur primarily without any previous tuberculosis of the lungs or intestines, on account of the tubercle bacilli passing by the lungs and intestines, without producing any tubercular changes in these organs. A primary tuberculosis of the uterus may spread through the Fallopian tubes to the peritoneum and through the diaphragm to the pleuræ. Primary peritoneal tuberculosis does not affect the parenchymata of the abdominal organs, with the exception of the uterus (Fallopian tubes) and the liver (portal region). It generally occurs in the anterior parts of the abdominal cavity, and then spreads in the direction of the lymph passages which proceed from the abdomen from rear to front. These lymph passages pass from the inferior and lateral abdominal wall, through the diaphragm, into the thoracic cavity. They first of all proceed to the posterior, then to the anterior mediastinal glands and to the bronchial glands, from which the pleuræ may also become tubercular. On the other hand, the process does not pass from the pleuræ to the lungs, with the exception of a small part of the *hilum pulmonis*. The primary tuberculosis of the pleuræ cannot spread directly to the peritoneum on account of the opposition offered by the lymph stream which flows from rear to front. The parenchyma of the lungs also remains free. Primary tuberculosis of the lungs ends either directly in tuberculosis of the pleuræ or at first attacks the lymph glands (bronchial glands) and from thence spreads to the pleuræ. Primary intestinal tuberculosis may also directly set up tuberculosis of the peritoneum or at first may affect the glands (mesenteric), and from thence proceed to the peritoneum. Besides this, primary intestinal tuberculosis may, without the instrumentality of the systemic circulation and without generalisation, lead to tuberculosis of the liver

if bacteria have penetrated through the portal vein into, but not beyond the liver. If tuberculosis becomes general from any of the above mentioned primary foci, the comparative implication of the organs which are affected in cattle are somewhat as follows : The lungs in 100 per cent. ; pleuræ and peritoneum in 90 ; liver in 85 ; oral and pharyngeal cavities, and the intestinal canal in 60 ; the spleen in 50 ; the kidneys in 30 ; and the bones in 5 per cent. In female animals, tuberculosis of the uterus is met with in 65 per cent. ; tuberculosis of the udder in from 5 to 10 ; and tuberculosis of the ovaries in 5 per cent. The sexual organs of males are much less frequently affected than those of females.

Rieck reports with regard to the statistics of the Leipsic abattoir (67,000 cattle), that in 80 per cent. of all cases of tuberculosis, the lungs and their glands were alone implicated, and that the disease was confined to the serous membranes and their glands only in 10 per cent. General tuberculosis was found in 3 per cent. The respective percentages in the various organs were as follows : lungs, 100 ; liver, 83 ; intestinal canal, 73 ; serous membranes, 57 ; kidneys, 52 ; flesh (muscles), 49 ; spleen, 18 ; udder, 17 ; and bones, 9. Rieck comes to the conclusion that the principal gate of entrance of the infection in cattle is the lungs, and that full-grown cattle are very seldom affected from the intestines.

Symptoms.—Tuberculosis of cattle is characterised by a very gradual development and by a very slow and chronic course. The initial stage of the affection escapes observation, and only in exceptional cases have signs of fever been noted as a first symptom. The symptoms vary according to the seat of the disease.

1. *Tuberculosis of the lungs* manifests itself, first of all, by a weak, dull, short cough, which, n the early stages of the irritation that gives rise to it, occurs in short jerks ; but, later on, in spasmodic and very distressing paroxysms. This cough is usually dry, and is seldom accompanied by expectoration. It is most severe in the early morning after getting up ; after exercise ; and after drinking. Respiration is more or less difficult and accelerated. With advanced destruction of the lungs, the animal pants, the ribs heave, and the nostrils remain dilated. Discharge from the nose is rare, as a rule. Sometimes, owing to dilatation of the bronchi, or breaking through of cavities of the lungs into the bronchi, a considerable quantity of a muco-purulent or caseous substance is discharged by coughing, in which cases the exhaled air has a bad smell. It often happens, if the sides of the thoracic wall or withers are pressed, that the animal will groan and cough. Percussion sounds are in many cases normal. If, however, tubercular changes spread over the surface of the lungs, and if they are sufficiently extensive, the percussion sound is dull on certain circumscribed spots, although usually

it is only weak. The vesicular respiratory murmur on auscultation is generally weak, or it may be altogether absent, according to the site of dulness. In other cases, it is sharper than usual; or we may hear râles (bronchitis), or, more rarely, bronchial respiration (cavities of the lungs and bronchiectasis).

If the disease has lasted for some time, the *state of nutrition* will usually be bad. The hair loses its gloss and becomes coarse; and the skin inelastic and hard to the touch, so that it can only with difficulty be pinched up into a fold between the finger and thumb ("hidebound"). The appetite gradually decreases, and digestive derangement ensues, on which account the secretion of milk diminishes very considerably. In some cases, we find the characteristic aspect of a chronic incurable flatulence (tuberculosis of the mediastinal glands with compression of the œsophagus). Other cases are characterised by increasing difficulty in swallowing, with salivation, nasal discharge, and shortness of breath (tuberculosis of the retropharyngeal glands), or dyspnœa with râles and stertorous breathing; extended position of the head and the neck, and increased sensibility and swelling of the larynx (tuberculosis of the larynx). Occasionally we find intermittent symptoms of colic, alternating with diarrhœa and constipation (tuberculosis of the intestines and tuberculosis of the abdominal glands); hæmaturia (tuberculosis of the kidneys); and a vaginal discharge (tuberculosis of the uterus). The symptoms are usually accompanied by enlargement and induration of the superficial lymph glands, especially the tracheal glands, subauricular glands (lymphomata of the parotid glands), upper, middle and lower cervical glands, glands of the shoulder, elbow, groin, hock, hip, udder, etc. The temperature of the body may be normal; although we far more frequently find an irregular, remittent, and even an intermittent fever up to 106° F., with a decided increase of temperature in the evening. The amount of blood in the body steadily decreases; the mucous membranes and the skin, the changes in which can be easily recognised in the neighbourhood of the udder and pudenda, become very anæmic; emaciation is excessive; the eyes are sunken; and the animal becomes increasingly languid and weak, and finally dies from diarrhœa or from general exhaustion. The course of the disease may last for months or even for years.

2. *Tuberculosis of the serous membranes* ("grape disease") in the majority of cases is not marked by any characteristic external signs. We may sometimes determine the presence of

tuberculosis of the pleuræ by the fact that a dull percussion sound is obtained over an extensive area, owing to the great development of the tubercles. Sometimes, but only in a few exceptional cases, we may hear a friction sound, which, according to our own experience, is by no means so common as is usually asserted. Tuberculosis of the peritoneum, when the genital organs are attacked, may manifest itself by peculiar sexual symptoms, which in cows are those of nymphomania. The animals frequently come on heat and usually remain so for an abnormally long time. They get very excited, mount their fellow cows, and do not become fecundated by taking the bull. Pregnant cows abort so frequently that abortion is in many cases the first symptom of pleural and peritoneal tuberculosis in a herd (Roloff). The general condition often remains good for a long time. We frequently find in this form of tuberculosis, the accessory symptoms mentioned in the description of tuberculosis of the lungs, especially emaciation and cachexia in the later stage of the disease. We may sometimes demonstrate the presence of peritoneal "grapes" on the rumen and on the portion of the peritoneum which lines the wall of the abdomen, by palpation of the hollow of the flank (Kleinpaul).

3. *Tuberculosis of the brain* may occur as an independent primary affection, and in most cases, is a complication of tuberculosis of the lungs. Its course is generally characterised by symptoms of an acute leptomeningitis. The animal shows great excitement and suffers from severe attacks of fury, convulsions and spasms, which often closely resemble those of cerebro-spinal meningitis, or which may have an epileptiform character. Later on, these symptoms alternate with unconsciousness, stupor and paralysis. It often happens that the patient collapses suddenly. In other cases we may observe symptoms of affections of the nerve centres, such as staggers (rotatory and circular movements); oblique carriage of the head (tuberculosis of the middle ear); paralysis of the facialis, oculo-motorius, opticus, and trochlearis; hemiphlegia; etc. Periodical attacks of excitement may sometimes be noticed. Tuberculosis of the spinal cord manifests itself by a heavy tread, high action, and finally by paralysis of the loins. The duration of the disease, according to Jensen, is from 2 to 6 weeks.

4. *Tuberculosis of the udder* may appear as a first and single symptom of tuberculosis in animals which are otherwise quite healthy, although it is more often secondary. According to

Bang, it manifests itself as a diffuse, painless, and comparatively firm swelling, usually of one quarter (one of the posterior quarters as a rule) of the udder; and more rarely, of two quarters. The milk, contrary to what is the case in other inflammatory conditions of the udder, is at first normal; but it becomes in about a month thin and watery, mixed with flakes, and sometimes, though not always, it contains bacilli. We ourselves have frequently searched in vain for bacilli in the milk of cows which were thoroughly infected with tuberculosis. Bollinger proved that milk from the udder of tuberculous cows was undoubtedly infectious in 55 per cent. of the cases; although the demonstration of tubercle bacilli in such infected milk succeeded only once in 20 cases. The swollen parts of the udder become more and more indurated, until at last they are as hard as a stone. The process frequently spreads from the posterior quarters to the anterior quarters of the udder. The supramammary lymph glands (pudic glands) often become enormously enlarged, and knobby on the surface. The diagnosis may be corroborated by intraperitoneal or intramuscular inoculation of the milk on guinea-pigs, and by "harpooning" of the udder (microscopic examination).

5. *Tuberculosis of the uterus* manifests itself, according to Hess, in a slimy, muco-purulent, ill-smelling vaginal discharge (patulous condition of the cervix, abortion or non-conception, and nymphomania). Also by the hard, knobby tumefaction of the uterus and sacral glands, which may be recognised on rectal examination. Besides, it is usually coincident with vulvar tuberculosis (lupus-like nodules and ulcers, as well as swellings), and always with general tuberculosis.

6. In *general tuberculosis*, the morbid changes described respectively under the previous five sections are combined in various ways. Besides, we may also find swellings of the bones and joints (*tumor albus*), stiffness and lameness (tuberculosis of the bones), as well as the before-mentioned tubercular changes of the skin and eyes. In very rare cases the disease suddenly assumes a peracute and fatal course (acute miliary tuberculosis). Finally, abortion is sometimes found to take place mostly in the seventh month.

Clinical Diagnosis, as compared to diagnosis by inoculation or by bacteriological methods, is very uncertain; because tuberculosis does not possess characteristic symptoms. No diagnostic sign may be present, especially during the first

few months of the disease. Even in the more advanced stages of the disease, an exact diagnosis from purely clinical data can seldom be made. This fact, which is denied without sufficient reason by some, is of great practical importance in forensic veterinary medicine; because, from a legal point of view, the proof of the existence of tuberculosis necessitates, as a rule, the slaughter of the animal. In connection with the tuberculin test, and with the bacteriological investigation and inoculation, a positive diagnosis of bovine tuberculosis can, in many cases, be made, during life, at the time of examination, and especially in most cases of tuberculosis of the udder, uterus and intestine. According to O. Müller, the diagnosis can at present be established with certainty in from 50 to 60 per cent. of the animals presenting clinical symptoms of sickness. In cases of pulmonary tuberculosis, on account of the highly perfected methods of examination, failures can seldom occur. On the whole, the following clinical factors are of importance in the event of a diagnosis, *intra vitam*, being required.

1. *For tuberculosis of the lungs:* The simultaneous occurrence of severe disturbances in nutrition, with symptoms of lung affection (coughing and the respective results of percussion and auscultation), and the enlargement of the external lymph glands, especially in cows and heifers. For securing the establishment of the rattling breath-sounds in the lungs, injections of arecolin and of pilocarpin have been recommended; also trotting of the animals before examination. The glands at the shoulder, bend of knee and udder, may also be cut out, without danger to the living animal, for the purpose of more certain diagnosis, and be examined on their divided surfaces (Ostertag).

2. *For tuberculosis of the serous membranes:* Increased sexual desire, sterility, tendency to abortion, friction sound (on rare occasions), and more or less extensive dulness. Here, perhaps, a knowledge of the breed may help us to arrive at a correct decision. The presence of tubercular proliferations of the peritoneum may be proved by introducing the hand into the rectum or by pushing the hand under the last false ribs. We may also mention incurable chronic flatulency with normal digestion.

3. *For tuberculosis of the brain:* Symptoms of acute inflammation of the brain, complicated with lung symptoms, glandular swelling, and loss of condition.

4. *For tuberculosis of the udder:* The highly characteristic.

at first diffuse, firm and painless swelling ; subsequent changes in the milk ; induration of the udder ; enlargement of the lymph glands ; proof of the presence of the bacillus in the milk ; the inoculation of the milk, and the "harpooning" of the udder. There are often present, besides, additional clinical indications of tuberculosis. On the other hand, the tuberculin test is not possible, additional tubercular foci being present. "Harpooning" of the udder is, according to Ostertag, best performed when the animal has been tied down. This operation is, when carried out aseptically, without danger even when repeatedly performed (with preliminary section of the skin and fascia of the udder). On the "harpooning" pieces of connective tissue tubercle may be recognised macroscopically—especially with the use of a pocket lens. The milk, in advanced cases of tuberculosis of the udder, is still virulent, even though diluted to the billionth degree (Ostertag).

5. Besides being aided by proof of chronic tympanites taken in connection with symptoms of pulmonary tuberculosis, our diagnosis of *tuberculosis of the mediastinal glands* will, according to Albrecht, be facilitated by the introduction of an œsophageal tube. He states that, immediately after the removal of the gas by means of this tube, normal digestion reappears for a shorter or longer period in cattle, contrary to what takes place in non-tuberculous cattle.

Up to the present, no practical experiments have been made with the ophthalmoscope for diagnosing tuberculosis of the choroid and iris. Röbert recommends, for facilitating the diagnosis, covering over, with a cloth, the nostrils and mouth of cattle which are being examined, in order to oblige them to adopt forced breathing, so that any abnormal respiratory sounds may become more easily heard. In a similar manner, an injection of 4½ grains of pilocarpin, by inducing forced breathing, will enable us to hear a well-marked friction sound in cattle suffering from tuberculosis of the serous membranes (Walther). Peuch states that we can make an exact diagnosis by inserting a seton and inoculating guinea-pigs with the produced pus. According to Wallmann, a slaughtered tubercular ox gave a distinct shadow with the Röntgen rays. He recommends this investigation also in cases of living animals.

Investigation of the Lymphatic Glands in Living Tuberculous Cattle.—Godbille, Director of abattoirs in Paris, made notable reports in the *Recueil* of 1895 on the methods of examining the lymphatic glands in living tuberculous cattle. The retro-pharyngeal glands were

most frequently found diseased. For palpation of these, the observer stands at the side of the neck; and, holding the thumb upwards, presses with the palmar surface of the fingers into the depths of the vicinity of the parotid gland, till the finger-tips of the two hands rest on the body of the first cervical vertebra. He can then move about the affected glands as knotty tumours (the normal size of the flat tongue-shaped glands is, in length, up to 6 cm., and in breadth up to 2 cm.=2.4 inch by 0.8 inch). Secondly, the sub-parotid lymph-glands are to be sought. For this purpose, the observer stands beside the neck, and places both hands under the corresponding ears in such a position that the ear-concha lies on the thumb and forefinger of the hand. Skin and subcutaneous tissue are now drawn forward in a single fold and pressed towards the lower jaw; whereupon an increase of size or a hardening of the two lymphatic glands, of normal dimensions (2.4 inches long by 0.8 inches broad), may easily be determined. The laryngeal glands are diseased only in exceptional cases. They do not lie in cattle, as they do in horses, on the inner surface of the jaw at the floor of the larynx, but at the margin of the lower jaw above the terminal tendon of the sterno-maxillary muscle. The comparatively rare diseased shoulder-glands are, in normal cattle, 7–10 cm. (2.8–4 inches) long; and may be moved under the skin by pressure of the finger at the anterior margin of the shoulder, just above the apex. In the diseased condition, they form spherical nodules. The glands of the knee-fold form strings of cylindrical shape and a small mass, 8–10 cm. in length (3.2–4 inches). They are placed in the fold of the abdominal cutaneous muscle, parallel to the anterior margin of the tensor fasciæ latæ muscle; and, in tuberculosis, are nodular and even enlarged to the size of apples. The supramammary (or retromammary) glands are normally of the size of a pigeon's egg. They are placed between the hind legs, above the posterior fourth of the udder, and may be readily moved to and fro by the hand. According to Ostertag, they may best be examined in the following manner. The observer moves the skin upwards on the lateral surface of the posterior fourth of the diseased side with the points of the index, middle, and ring fingers, to the fold of the thigh, and then feels with the corresponding fingers for the posterior margins and lateral surfaces of the lymphatic glands.

Bacteriological Diagnosis.—The bacteriological demonstration of the tubercle bacillus does not possess the great importance in bovine that it does in human tuberculosis, as it cannot practically be produced in many of the cases. The chief reason of this lies in the fact that in cattle, a tubercular lesion of the lung is not always furnished with tubercle bacilli, as in man; these being present only in a certain small percentage of the diseased animals. For the same reason, the method proposed by Pöls, of obtaining mucus for bacteriological examination, by inserting a cannula between two of the tracheal rings, is frequently without result. For instance, in the case of a cow which was suffering from fully-developed tuberculosis, we succeeded by this procedure in

obtaining bacilli only after repeated experiments; and in only one single streak preparation were able to demonstrate the presence of a few tubercle bacilli. Nocard's plan of obtaining mucus from the trachea, by injection of veratrine or eserine, is equally uncertain. Ravenel has lately recommended, during coughing, the application of a feeding-bag with a piece of wood placed at the bottom of it. It is frequently so difficult to find the bacillus, even in sections of the tubercles, that a large number of preparations have almost always to be examined. According to Gaffky, the bacteriological examination of the dung will furnish important diagnostic data.

Milk gives better results for bacteriological examination. But it is only in cases of tuberculosis of the udder that it contains tubercle bacilli which are microscopically recognisable by staining. In all other cases, and more especially in pulmonary tuberculosis and in grape disease, the milk usually contains no tubercle bacilli; even in cases of generalised tuberculosis, when the blood contains very numerous bacilli, they are absent from the milk (Ostertag, Bang, Nocard). The milk of cows which display no clinical phenomena (especially those which react to tuberculin), much more rarely contain bacilli. The testing of bacilli-holding milk by spread-out preparations is particularly uncertain. Bollinger found that among 20 tuberculous cows the milk was infectious in 11 cases; but that the presence of the bacilli could be microscopically proved in only one of them. Beyond the procedure of "harpooning" of the udder, recommended by Nocard, Ostertag, and McFadyean, with conclusive macroscopic and microscopic examination of the gland preparations thereby obtained—which does not always afford decisive results—the only sure means is the inoculation of experimental animals, with the bacilli-holding milk (and with other suspected matters, such as the vaginal secretion, uterine discharge, and extirpated lymphatic glands).

According to the experiments of Arloing, Bollinger, Verneuil and others, guinea-pigs give better results for this purpose, than rabbits. For inoculation, it is best to introduce a minute quantity of the infectious material into the abdominal cavity of these animals, when generally an abdominal tuberculosis with characteristic anatomical and microscopical conditions will develop after 3 or 4 weeks (multiple tuberculosis in the omentum, spleen and liver, as well as on the parietal peritoneum). Or we may, according to Ostertag, employ more advantageously the intra-muscular inoculation (muscles of the internal and

posterior surfaces of the hind-limb), as a decision is sooner rendered possible in this way than by intraperitoneal inoculation; because often, even within 10 days after the intramuscular inoculation, the neighbouring lymphatic glands may be distinguished as hard, painless nodules of the size of small peas. Experimental inoculation of guinea-pigs, with microscopic identification of the bacillus, must, accordingly, at the present time, be regarded as the surest diagnostic means of recognising tuberculosis in living animals. This inoculation is essential in all the cases in which the bacteriological diagnosis is doubtful (acid-fast pseudo-tubercle bacilli).

Microscopic Demonstration of the Tubercle Bacillus.—1. According to Ehrlich and Koch, the material to be examined (mucus, milk, pus, etc.) should be spread out into the finest possible film upon a cover glass. In the case of milk, Johne advises that it should be diluted with from 2 to 5 times as much water, and mixed with sufficient acetic acid to precipitate the albumen and bacilli. The precipitate is then placed on the cover glass, and is allowed to dry in the air, after which the cover glass is passed several times slowly through a gas flame. As soon as it is cool it is floated, with the film side downwards, upon the cold staining solution, which is described in detail further on, and in which it is allowed to remain from 12 to 24 hours. The cover glass should remain in the cold staining solution this length of time, and not, as formerly stated, from half to one hour. A better plan, as we have found, is to place the cover glass not merely in the staining solution, but to heat both together over a gas jet until perceptible steam is given off; and after they have been allowed to cool, to repeat the process once or twice in a similar manner. By this more expeditious method we obtain better preparations. The staining solution is made in the following way: 1½ f. drams of aniline oil are mixed with 3½ oz. of distilled water and filtered. To 3½ oz. (100 c.c.) of this aniline water are added 3 f. drams of an alcoholic solution of methyl violet, or fuchsin, and nearly 3 fluid drams of absolute alcohol. This solution should be renewed every 10 or 12 days. After the cover glass has been taken out of the staining solution, it should be rinsed in water and placed for a few seconds in dilute nitric acid (1 to 3), by which it is decolorised, and it must then be again thoroughly rinsed in water. By means of this method of decolorisation, the tubercle bacillus alone retains the stain. The cover glass is finally put for about five minutes in a 1 to 2 per cent. watery solution of Bismarck brown or malachite green, rinsed, dried, placed in Canada balsam, and put under the microscope. By this method the tubercle bacilli are stained dark-blue or red, whilst all other bacteria and cells appear brown or green.

2. Gabbet has published a new staining method, which, he states, is an extremely convenient one in practice for rapid preparation. He places, for two minutes, the dry preparation, which is made in the usual way, in a fluid composed of 3¼ oz. of a 5 per cent. aqueous solution of carbolic acid and ¼ oz. of absolute alcohol, in which 15½ grains

of fuchsin has been dissolved. Directly afterwards he puts it for one minute in a mixture of 3¼ oz. of a 25 per cent. solution of sulphuric acid, in which 31 grains of methylene blue have been dissolved. It is next rinsed in water, and examined in this medium or still better it is rinsed in absolute alcohol and mounted in Canada balsam. The bacilli will then appear red on a blue ground. If a more intense staining of the bacilli be required, the first solution is heated for two minutes until steam arises from it.

3. According to Ziehl-Nelsen, the preparation to be examined is placed in the usual way upon a cover glass, is passed through the flame, and is then immersed in a solution of carbolic acid and fuchsin, which is heated over a Bunsen's burner in a hollow ground slide on a ring of wire netting. The said solution consists of 1 part of fuchsin, 5 parts of carbolic acid, 10 of absolute alcohol, and 95 of distilled water. As soon as the solution begins to give off steam, the preparation is taken out and placed first of all in a 5 per cent. solution of sulphuric acid, then in a 70 per cent. solution of alcohol, and finally in a solution of 2 parts of methylene blue in 100 parts of water. The preparation is then washed in water and placed upon the slide.

4. Czaplewski moistens with carbol-fuchsin the previously prepared cover glass preparation, which has been got ready in the usual way, and holds it over the flame till it gives off vapour. On draining away the carbolic fuchsin solution, the cover glass is dipped from six to ten times into a concentrated alcoholic solution of yellow fluorescine. It is then dipped ten or twelve times in a concentrated alcoholic methylene blue solution, and finally rinsed in pure water.

With regard to other staining methods (Weigert, Löffler-Fränkel and others), consult the text-books of bacteriology.

Diagnostic Inoculations in Guinea-pigs.—Intramuscular inoculation of the muscles of the internal and posterior surfaces of the hind-limb give the readiest results; the neighbouring lymphatic glands are often altered in 10 days into hard, painless nodules of the size of small peas. The most frequently used are, however, intraperitoneal inoculations. Numerous tubercles of the size of a millet seed form in from 3 to 4 weeks on the peritoneum after the injection. Subsequently we may find many caseous tubercles varying in size from a pea to a bean, in the omentum and in the enlarged spleen; and small yellow nodules in the liver. If a subcutaneous inoculation be made, a nodular induration will develop at the site of the injection. In the second week, a suppurating ulcer forms, and from the second to the third week the neighbouring lymph glands swell to the size of a hazel nut. After the sixth week, marked emaciation takes place. If a *post-mortem* examination be made from 6 to 12 weeks after the inoculation, we shall find that the tubercles at the site of the injection and the neighbouring lymph glands are caseous. Numerous tubercles and caseous foci are found in the mesentery, omentum, liver, and in the greatly enlarged spleen. Occasionally miliary tubercles become developed in the lungs.

Agglutination procedure.—The diagnostic significance of the agglutination methods introduced by Arloing-Courmont have been very

unfavourably viewed, out of France; while the serum-reaction is characterised as wholly unreliable (von Behring, Beck, Lydia Rabinowitsch, and others). The diazo-reaction of Ehrlich is also unfavourably regarded as a diagnostic agent in bovine tuberculosis (Klimmer and Schmidt).

Pseudo - tubercle - bacilli.—In recent years there have been found, especially in milk and in butter, bacilli which greatly resemble tubercle bacilli (especially the acid and alcohol resisting bacilli), and produce in inoculated animals (guinea-pigs) processes similar to those of tuberculosis in the peritoneum and abdominal organs. These are known as soil-bacilli and grass-bacilli (Lydia Rabinowitsch, Petri, Möller, Korn and others).

Tuberculin as a Diagnostic Agent.—Tuberculin, which was discovered by R. Koch in 1890, and which is a glycerine extract of pure cultivations of tubercle bacilli, has, in human medicine, proved to be neither a remedy nor a diagnostic agent. On the other hand, the results of numerous inoculations of tuberculin in cattle demonstrate the fact that, tuberculin is a very valuable, although not an absolutely infallible aid to the diagnosis of bovine tuberculosis. From experiments made in many places, we may deduce the following rules for tuberculin inoculations:—The dose of tuberculin for horned cattle is 8½ mins., for calves 1½ mins. It is injected under the skin, diluted with ten times its bulk of a watery solution of carbolic acid of ½ per cent. Smaller doses are sometimes sufficient, although the reaction they produce is often too weak and transient. The reaction in tuberculous animals consists chiefly in a rise of temperature (inoculation fever or reaction fever) which appears in from 12 to 15 hours after the injection of the tuberculin. If an increase of temperature of at least 1·8° F. is found in an animal after the inoculation, the presence of tuberculosis may be assumed as probable. If, before inoculation, the temperature of the bovine patient be abnormally high, especially if it be 103.1° F. or more in the rectum, no reliable deduction can be made. We must also point out that the reaction, as a rule, is inversely proportionate to the extent and duration of the disease, so that excessively tuberculous cattle show less increase of temperature than those with slight localised tuberculosis. Hence, in severe and old standing cases of tuberculosis, tuberculin is less reliable. Experience has also shown that animals, which have been previously inoculated with tuberculin, react but slightly, or not at all, to injection of the ordinary dose (they reacted in the Prussian marine quarantine to a dose of five times the quantity). One

inoculation generally suffices. In doubtful cases it may be repeated after a few weeks. The evening is the best time to make the inoculation; for then the rise of temperature can be observed during the following day. The temperature must be taken at least four times after the inoculation, namely, 9, 12, 15, and 18 hours after injection, prior to which the temperature should be taken at least twice, that is to say, six hours before, and directly prior to the injection of the tuberculin. The side of the neck is the best site for the inoculation. Tuberculin has, only in a very few cases, caused injury to the inoculated animals (acute miliary tuberculosis, fatal intestinal hæmorrhage, œdema of the lungs, temporary sterility, and contraction of the uterus). The quantity of the milk becomes diminished during the time of the reaction, to the extent of a few quarts, in consequence of the resulting fever and loss of appetite.

In a great number of cases, cattle which had reacted to tuberculin have, on being subsequently slaughtered, proved free from tuberculosis. Such failures in diagnosis have been estimated at 13 per cent. in Germany and at 10 per cent. in Denmark. Cattle suffering from the following diseases sometimes react in the same way as those that are affected with tuberculosis: actinomycosis, botryomycosis, abscesses of the lungs and liver, abscesses following traumatic pericarditis, inflammation of the udder, caseous echinococci, distomatosis, diseases due to pulmonary worms, emphysema of the lungs, and chronic diarrhœa. As it is not a specific diagnostic agent for tuberculosis, its reaction cannot be accepted in forensic veterinary cases as a positive proof of the presence of tuberculosis; although the fact of the reaction occurring would strongly suggest such a probability. On the other hand, an animal may, under certain circumstances, be tuberculous even when the alteration of temperature does not quite reach a whole degree. Tuberculin, however, may be used with advantage for diagnostic purposes, when we wish to select animals for breeding, and as a prophylactic for combating tuberculosis in large herds.

The *Prussian Ministerial Instructions of October 29, 1900*, decide that:

(*a*) Those cattle must be regarded as reacting, and suspected of tuberculosis, which have not shown a temperature above 103° F. before inoculation, but where the body-heat ascended after the injection of tuberculin above 103° F.; when the difference between the highest point of temperature recorded before and after the injection has been at least 1.8° F.

(*b*) In calves, up to the age of 6 months, a rise of the internal temperature of the body above 104° F. after injection of tuberculin gives grounds for suspicion of tuberculosis; if the alteration of temperature has reached at least 1.8° F.

With regard to *Quarantine regulations*, the Ministerial order of May 20, 1901, directed the official presidents in Schleswig as follows:

Cattle which have, before the inoculation with tuberculin, already displayed a febrile rise of the internal temperature, are to be carefully examined as to whether such symptoms of sickness are also present, which, in association with the rise of temperature, would give clinical grounds for suspicion of tuberculosis. Animals in which such symptoms exist, are not to be inoculated, and are to be treated as clinically suspected of tuberculosis, and, accordingly, sent back. In the remaining cases (older cattle with a temperature over 103° F.; calves up to 6 months of over 103° F., as the result of fatiguing journeys or some other cause), tuberculin inoculation is to be carried out; and with the provision that those animals are to be regarded as reacting, in which difference between the highest temperatures before and after the tuberculin injections, respectively, reached to 0.9° F. and upwards.

Differential Diagnosis in Cattle.—1. Tuberculosis of the lungs may be *clinically* mistaken for disease of the lungs due to echinococci, after-conditions of pleuro-pneumonia, and verminous bronchitis. Tuberculosis of the serous membranes may be mistaken for pleuritis; tuberculosis of the mediastinal glands, for ordinary chronic tympanitis; and tuberculosis of the uterus, for chronic metritis. Tuberculosis of the brain is frequently mistaken for simple inflammation of the brain, cerebro-spinal meningitis, rabies, malignant encephalitis (Zündel), staggers, etc. Tuberculosis of the lymph glands may also be easily mistaken for actinomycosis, leucæmia, and general sarcoma. There is no doubt that many reported cases of tuberculous lymphomata of the pharyngeal cavity, region of the parotid glands and oral cavity, and of tuberculosis of the tongue and udder, were nothing else than actinomycosis. Tubercular arthritis and tendo-vaginitis have usually been confounded with pure surgical paralysis or with rheumatism, and tuberculosis of the shoulder glands, with lameness at the shoulder. A complication of tuberculosis and actinomycosis sometimes occurs. In the majority of such cases, no positive conclusion can be arrived at without making a section for microscopical examination.

2. *Anatomically*, the following diseases may be mistaken for tuberculosis: Pleuro-pneumonia contagiosa (the lungs); actinomycosis (mouth, pharynx, lymph glands, udder, and

bones); leucæmia (liver and kidneys); amyloid degeneration, (liver, spleen, and kidneys); metastatic abscesses and caseous echinococci and cysticerci (lungs, liver, spleen, kidneys, lymphatic glands, subserous tissue); broncho-pneumonia; pneumonia due to foreign bodies; bronchitis; peribronchitis; gastro intestinal ulcers; suppurative endometritis, sarcoma, multiple calcification of the peritoneum. The mistakes which most frequently occur in practice are those with caseated echinococci, calcified pentastomata, and actinomycosis. The distinction is, as a rule, easily made by microscopic methods (actinomycosis kernels, lamellar structure of echinococci, cuticula). Tuberculous foci are also characterised as such by the coincident disease of the lymphatic glands of the vicinity (nodules, caseation, calcification), and by the occurrence of small nodules in the neighbourhood of the larger ones.

Special attention has been lately directed to the discrimination between tuberculosis of the liver and echinococcus multilocularis. The disease, set up by echinococcus m., is distinguished, according to Ostertag, from tuberculosis by the fact that it does not cause a simultaneous affection of the corresponding lymph glands; by the affected part being elastic; and, as a rule, by the eruption of fresh vesicles on its border.

Prognosis.—Tuberculosis in cattle is curable only during the very early stages and when it is local. In all cases, the prognosis is very unfavourable, and consequently it is best to slaughter the animal as soon as possible.

Therapeutics.—It is futile to treat tuberculosis by medicine. Even tuberculin appears to possess no curative influence. Whether cinnamic acid (which is said to have been used with good results in human tuberculosis) and iodipin (which is recommended as a specific in bovine tuberculosis), will prove themselves to be possessed of any curative power, is doubtful. Therapeutics must, accordingly, restrict itself to the employment of certain prophylactic measures, in which connection, the veterinary police regulations take precedence.

1. The **Veterinary Police** regulations are restricted to combating the clinically recognisable forms of tuberculosis (advanced cases of tuberculosis of the lungs, udder, uterus, and in-

testine). These forms have been made legally notifiable, and subject to veterinary investigation. Segregation and slaughter have been legally prescribed for cattle affected with any of the clinically recognisable forms of tuberculosis. In view of the danger of the distribution of tuberculosis by milk (especially among pigs), orders must be given to all dairies that the milk is to be delivered to the purveyors only after being heated to 185° F. The owners of cattle condemned to slaughter must be indemnified from a tuberculosis fund levied in the neighbourhood, and maintained at a definite government rate, since the campaign against tuberculosis is, above all things, carried on in the public interest.

That tuberculosis of cattle can be successfully combated by these regulations, the experiments for its extermination, which were carried out by Ostertag in East Prussia, have practically demonstrated.

2. **Protective inoculation** ("Jennerisation") has recently (1902) been recommended by von Behring for the purpose of combating bovine tuberculosis. According to experiments carried out in Marburg, dried bacilli of human tubercle produced, by intravenous inoculation of healthy cattle, an increased power of resistance to subsequent artificial infection. Von Behring thinks that a single inoculation suffices for immunisation against the natural infection.

The "Jennerisation" is carried out in this way: The newborn calves receive the colostrum of the cows for the first two days, and are afterwards fed with sterilised milk. At the age of from 4 to 6 weeks, the first intravenous inoculation (jugular vein) is carried out, of 0.004 grams (0.06 grains) of tubercle bacilli in 4 grammes (1 drm.: a teaspoonful) of water. If a febrile reaction results, the single inoculation is sufficient; in other cases, a second inoculation, with 25 times the quantity of tubercle bacilli (0.01 : 4), is carried out.

Whether von Behring's procedure is to be recommended for general adoption is a question to be decided by extensive practical research. It must in this way be proved whether the inoculation is without danger to the calves; or if inoculation with fully virulent living bacilli does not convey with it some danger to the non-immunised cattle, as well as to other domestic animals, and even to human beings; and if equal results are to be obtained from the unequal virulence of individual tubercle bacilli.

Extinction of Tuberculosis according to Bang.—The method introduced by Bang for the extinction of tuberculosis in Denmark is as follows: 1. All calves are separated on the second day after birth (on the first day the colostrum is indispensable to life) and fed on boiled milk only. Some weeks after birth they are inoculated with tuberculin, whereupon those which react are eliminated. 2. All the cattle of a shed are inoculated with tuberculin. After disinfection of the shed, it is divided into two parts by a partition. The healthy animals are placed in one division, and the reactionary (sick) in the other. Separate attendants are appointed for each division. The tuberculous animals are slaughtered as soon as possible. Tuberculin inoculation is carried out on all cattle twice a year. In Prussia, the experimental application of Bang's procedure, which has been made in many farmyards and districts, has not proved satisfactory; while the regulations laid down, especially that of the introduction of a partition, have been found almost impracticable.

Legal Instructions as to the disposal of the Flesh of Tuberculous Animals.—In § 34–40 of the law of Germany of 1900, with reference to the Government inspection of meat, there are the following fundamental rules for the determination of the fitness of the meat for food. § 33—In tuberculosis the whole of the carcase (flesh with bones, fat, viscera and blood) is to be looked upon as unfit for human food and injurious to health, when the animal, as the result of the disease, has reached an extreme degree of emaciation. § 34—Also (the fat only being excepted) when even without extreme emaciation, the signs of a recent blood infection are present, and when these are not confined to the viscera and the udder. § 35—Also those parts only of the flesh in tuberculosis with exception of the cases of § 33 and § 34. An organ is also to be regarded as tuberculous if only the associated lymphatic glands display tubercular changes; the same rule holds good of pieces of flesh when they are not proved free from tuberculosis on precise examination. § 37—As appropriate for use must be regarded: (1) the fat in cases of § 34; (2) the whole quarter of flesh in which a gland is found that has undergone tubercular alteration, unless it is to be regarded as unfit for use according to § 35; (3) the whole body of the animal, with the exception of those parts which are to be considered as unsuited to use according to § 35, when tuberculosis is present that is not confined to a single organ, and has not produced an extreme degree of emaciation, and either (*a*) extensive masses of softening are present, or (*b*) signs of a fresh blood infection are evident, but only in the viscera or the udder. § 38—The flesh which has been recognised as conditionally fit for use, is to be made suitable for human consumption; the fat by melting, in the cases referred to in § 34; and the flesh and the fat by cooking or steaming in the case of tuberculosis referred to in § 37.

Forensic Aspect.—In Germany it is set forth by the new Municipal Law Code that in cases of tuberculosis: (1) in the sale of animals, for general use and for breeding purposes, the requirement is a warranty of 14 days; (2) in the sale of animals for slaughter, in the case of cattle and of pigs, a warranty for 14 days is also required.

2.—Tuberculosis of Pigs.

General Remarks.—Tuberculosis of swine, especially in young pigs, is a fairly common disease, although it is far less frequent than tuberculosis of cattle. In the abattoirs of Germany the number of tuberculous pigs is from 2 to 6 per cent. and is yearly increasing. In Prussia, the percentage has steadily increased of recent years, from 1 per cent. in 1891, to 1.4 in 1895, 1.9 in 1896, 2.1 in 1897, 2.3 in 1898, 2.8 in 1899, and 2.4 in 1900. In Saxony the percentage was 1.0 in 1891, 2 in 1894, 2.2 in 1895, 2.7 in 1896, 3.1 in 1897, 3.2 in 1898, 3.03 in 1899, and 3.5 in 1900. In Bavaria in 1896, the percentage was 0.22, 0.26 in 1897, 0.35 in 1898, 0.41 in 1899, and 0.46 in 1900—of all slaughtered pigs. In Berlin, of 3,500,000 pigs slaughtered in the years 1883 to 1892, 56,000 were found tuberculous (=1.6 per cent.); in 1892, the percentage was 2.7; in 1896, 3.1; in 1897, 3.5; in 1898, 3.9; and in 1900, 4 per cent. In the years 1895 to 1900, the percentages of the slaughter-houses were, in Dantzic, 11; Kiel, 6–6.5; Zwickau, 6–7; Freiberg in Saxony, 7.7; Gera, 4; Königsberg, 4; Bromberg, 3.5; Leipzig, 3; Magdeburg, 2–3; Hanover, 1–1.3. In Denmark from 10 to 14 per cent. of all the slaughtered pigs are tuberculous. Tuberculosis is said to be very common among pigs in Belgium. In England, the proportion in 1896 was calculated at 1 per cent. In Sardinia, tuberculosis of pigs appears seldom to occur (Sanfelice).

Among pigs, which thus differ from cattle, tuberculosis is found to be most common in those that are less than 1 year old. The predisposition to the disease is greatest in improved English breeds. The chief causes which act unfavourably on the constitution of swine are: unnatural rearing, fattening, exclusive stall-feeding, and early forcing. The commoner the breed, the less liable it is to tuberculosis. Hertwig points out that the pigs of Servia, Galicia, and the Bakony forest (Hungary), which have been but little improved in breed, seldom become tuberculous; probably because most of them are pasture-fed. The chief cause of tuberculosis of pigs, and that of its steady increase, is to be found in the communication of bovine tuberculosis in feeding with the raw offal and refuse of dairies, and especially the crude sediment left from the separator. The ingestion of the tubercle bacilli frequently takes place through the milk of tuberculous sows, when all the members of a litter may become affected. The respiratory air must also be

considered to be a vehicle for the infection; because experience proves that all the animals of a sty often become simultaneously tuberculous. As in cattle, we must assume the heredity of tuberculosis in swine; because all the pigs of certain breeds become affected by the disease. Meyer, Michael, Tempel, and others have remarked that in castrated pigs, infection may originate in the operation wound. It has not yet been proved, although it is probable, that infection may be transmitted to pigs by the tubercular sputum and saliva of man.

Pathogenesis and Anatomical Conditions.—Tuberculosis of pigs differs from that of cattle principally by the fact of its being a well-developed food-tuberculosis; as its starting point is generally in the digestive apparatus, and bacilli can only with difficulty be found in the affected organs. According to Ostertag, tuberculous inflammation is met with chiefly in the jejunum, ileum, and cæcum, with ulceration of their mucosa, and formation of tubercles in the muscularis, subserosa and mesenteric glands. At the same time, we may find tubercular tonsilitis (less frequently pharyngitis) and a diseased condition of the submaxillary, retropharyngeal, and upper cervical lymph glands. The encroachment of tubercular intestinal ulcers on a branch of the portal vein causes embolic tuberculosis of the liver; in the event of an invasion of numerous bacilli, extensive miliary tuberculosis of the liver, and consequent general tuberculosis. The tuberculosis of the middle and inner ear is of special importance. According to Schütz, it begins with catarrh of the pharynx, in which case the tubercular inflammation at first spreads through the Eustachian tube to the tympanic cavity and sets up a tubercular osteomyelitis, periostitis, and parotitis with rarefaction and necrosis of the bones. From thence the process may extend to the cerebral membranes, the cerebellum, medulla oblongata, and even into the external auditory passage, which becomes filled up by a tubercular tumour.

When, as happens in rare cases, swine tuberculosis begins primarily in the respiratory apparatus, it takes the form of a tubercular broncho-pneumonia, chiefly at the base and apex of the lungs, with consequent implication of the bronchial glands, and, less occasionally, of the pleuræ. In many localities, inhalation-tuberculosis appears to occur as frequently as food-tuberculosis, *e.g.*, in Hanover (Ströse). The lungs, especially on their surface, become studded with numerous miliary and sub-

miliary, grey, translucent tubercles, or with larger hard tubercles which vary in size from a pea to a hazel-nut, and which undergo caseous disintegration in their centre, and gradually become indurated, being in some cases as hard as a piece of cartilage. The surface of the lungs thus presents a spotted and uneven appearance. Besides this, we may find comparatively large caseous pneumonic foci, the formation of interstitial tissue, and chronic bronchitis, with their results. In consequence of the diseased condition of the pleuræ, the mediastinal glands and the subpleural lymph glands of the thoracic vertebræ and sternum may also become infected. We rarely see tuberculosis of the trachea and larynx along with tuberculosis of the upper, middle, and lower cervical glands. Primary tuberculosis of the uterus is also rare. On the other hand, general tuberculosis is common in pigs.

Statistics.—Ostertag states that the lungs in general porcine tuberculosis become affected in 100 per cent. of the cases; liver, in 90; spleen, in 80 to 85; oral cavity, laryngeal cavity and intestinal canal, in 80; kidneys and serous membranes, in 30; bones, in 15 to 20; mammæ, in 5 per cent.; testicles, uterus, and articulations, in 1 per cent. This table shows that in pigs the spleen is much more frequently, and the serous membranes of the abdomen and thorax far more rarely affected by tuberculosis than in cattle. Ströse found generalised tuberculosis in 328 instances; local tuberculosis of lungs or bronchial glands in 148; while in 48 the tuberculosis was limited to the mesenteric lymphatic glands, intestine, upper cervical glands and liver. Lungwitz observed 141 cases of tuberculosis of serous membranes; generalised tuberculosis was associated with it in 134 cases.

Symptoms.—The symptoms of tuberculosis in pigs vary according to the part affected. Frequently there is no external sign of the disease.

Young pigs when suffering from *intestinal tuberculosis* are backward in growth and become gradually emaciated. The mucous membranes are very pale, and the skin gets covered with dark grey crusts (*Russ der Ferkel, suie des porcelets*, soot of young pigs). These symptoms are followed by various disturbances of digestion, such as vomiting, tympany, slight colic, and diarrhœa. The internal temperature is alternately high and normal. Later on, the patient steadily loses condition. The flanks are tucked up, the eyes sunken, and the belly pendulous. The adherent intestinal loops can frequently be felt from the outside as a hard knotty mass of the size of a man's fist, or even larger. The animal sometimes manifests pain during palpation

of the posterior part of the body. Death occurs from progressive general debility, usually in a few months, or even weeks, if the lungs and intestines be simultaneously affected.

Tuberculosis of the brain in pigs runs a very irregular course. We first observe automatic movements, such as rearing and turning round and round; convulsions and spasms of the muscles of the trunk and limbs; nystagmus; etc. At a later stage the following paralytic affections appear: hemiphlegia, unilateral paralysis of the tongue, oblique position of the head, paralysis of the muscles of the eyes, and especially of the eyelids; unilateral paralysis of the face with the snout drawn to one side; and pendant condition of the ears. Tuberculosis of the vertebral column leads to lumbar weakness and paralysis of the hind quarters.

In *tuberculosis of the lungs* in pigs, the sufferer has at first a short and dry cough, which becomes, later on, very harassing, and generally with choking paroxysms. The breathing is accelerated and forced, and the animal becomes gradually emaciated. The disease has a shorter course in young pigs than in old ones, and may last for weeks or even months.

The chief *diagnostic points* are: Occasional visible tubercular proliferations in the outer auditory passage; swelling of the cervical lymph glands; and eruption of tubercles in the interior of the eye (Azary). Bacteriological examination is of less importance for diagnosis in pigs than in cattle; because even in anatomical preparations, the bacilli can be found only with the greatest difficulty in swine, especially in the caseated and calcified masses. In section preparations they are more easily identified (Olt). On the other hand, we can have recourse to a diagnostic tuberculin inoculation (according to Thiro, the normal temperature in young pigs registers an average of 104° F.; in the adult ones 102° F.); while the inoculation dose is 0.1–0.3 grm. (1½–5 mins.) of tuberculin.

Differential Diagnosis.—As tuberculosis in pigs can, as a rule, be proved only after slaughter, we should above all things pay attention to the anatomical changes, which we should be careful not to mistake for the chronic processes of contagious pneumonia of the pig. Ostertag differentiates tuberculosis and contagious pneumonia as follows:—

1. In *tuberculosis*, changes may be found in all the organs of the body. Solid tubercles along with caseous foci may be met with in the parenchyma. The lymph glands of the tubercular

organs are invariably enlarged, and caseation in them always emanates from several points, which correspond to the conglomerates of the tubercles, so that the enlarged lymph glands appear to be infiltrated by numerous small caseous foci. As a rule, calcification quickly follows caseation.

2. In *chronic* pneumonia, changes are generally found in the lungs, and, less frequently, in the skeleton. We find in the lungs only cavities or formations of sequestrum, varying in size from a pea to a man's fist. Usually an adhesive pleuritis is also present. The lymph glands most frequently affected are the bronchial, tracheal, and inguinal glands. When attacked, they become entirely caseous and present the appearance of a round sack filled with dried caseous pus, arranged in concentric layers without any calcification. The bacteria of contagious pneumonia can be easily found in the pus foci.

It is worthy of remark that tuberculosis and chronic pneumonia may occur simultaneously in a pig, in which case the presence of tuberculosis may be proved by experimental inoculations in guinea-pigs.

Therapeutics.—In view of prophylaxis it is specially to be recommended that all refuse should be burnt, and all the skimmed milk and butter milk from the dairies should be sterilised by heating to 185° F. In other matters, the same general hygienic rules should be observed as in bovine tuberculosis.

Scrofulous or Caseous Inflammation of the Intestines.—The etiology of the disease of pigs which was described by Roloff under this name, has not yet been properly elucidated. It probably belongs partly to tuberculosis, and partly to swine fever, and is characterised by peculiar intestinal changes that chiefly implicate the colon, the loops of which form, by adhesion, a firm bundle. The individual intestinal convolutions represent thick eminences with divisions and cavities by which the intestine assumes a rosary-like aspect. The wall of the intestine is thickened and covered with nodular prominences, the centre of which contains caseous foci. Miliary nodules are imbedded under the serosa and the submucosa. The mucous membrane of the intestine shows circumscribed, copper-coloured hæmorrhagic foci or necrotic disintegration, with scabbing and exfoliation of the upper layers, and sometimes has broad caseous masses on its surface. Peyer's patches are swollen and ulcerated. Large tubercular ulcers, with raised and thickened margins, are found in the mucous membrane. The ileo-cæcal valve frequently projects into the cæcum in the form of a long firm plug, with a scabby discoloured surface and ulcerated margin. The intestines become sometimes narrowed by cicatrisation. The mesenteric glands are enlarged and contain caseous foci.

3.—Equine Tuberculosis.

Occurrence.—Tuberculosis is not a common disease in horses. Many of the cases which were published long ago are not altogether free from suspicion. On the other hand, it is not improbable that a few cases reported as glanders were really those of tuberculosis. In recent times, the presence of tuberculosis has been demonstrated more frequently in the horse than formerly. Bang has, for instance, collected 29 cases. In the Prussian army, 10 horses died of tuberculosis in the years 1894 to 1898. In the abattoirs of Prussia, 0.1 per cent. of the horses slaughtered in 1899 and 1900 were found to be tuberculous. In Saxony 0.08 per cent. of 3,500 slaughtered horses were found to be tuberculous ; in 1895, the percentage was 0.09 ; in 1896, 0.34 ; in 1897, 0.33 ; in 1898, 0.16 ; in 1899, 0.17 ; in 1900, 0.22.

Pathogenesis.—The chief gate of entrance in horses for the tubercle bacilli is the lungs ; a fact which may be inferred from the frequency with which these organs participate in the disease. Also, the affection in them is often primary (Nocard and Johne). Nothing certain is known as to the source of the infection, whether it be horses, cattle or men. In some cases, transmission from cattle through cohabitation appears to take place. Csokor and Bang consider that the intestines are one of the chief points of invasion for the bacteria. Their entrance along with the food is proved by the fact that the oldest and most important tubercular changes are found in the mesenteric glands. Bang states that the bacilli, among other things, are transferred to horses by feeding with the milk * of tuberculous cows

* McFadyean (*Journal of Comparative Pathology and Therapeutics*, September 1896) draws attention to the fact "that in a considerable proportion of cases there was a distinct history of the animal having been largely fed with tuberculous milk. Now when one reflects that certainly not one horse in several hundreds is at any period of its life fed on cows' milk, the frequency with which tuberculosis has been met with in horses that had been so fed, becomes very striking." It is obvious that if we give milk to horses, either in health or disease, we should sterilise it in the event of our not being certain that the cows from which the milk was taken were free from tuberculosis. In sterilising milk by heat, we should be careful, if possible, not to allow it to attain a higher temperature than, say, 194° F. (taking the death-point of the tubercle bacilli at 187° F., see Vol. II., p. 332), so as to avoid imparting to the milk the peculiar taste which boiling gives it, and which is disagreeable, as a rule, to horses. Allowing it to get even in the slightest degree smoked, renders it still more unpleasant to the would-be equine drinker.—Tr.

and by the use of straw from infected cow-sheds. Reimsfeld has also observed the occurrence of tuberculosis in a foal, after being fed with unboiled skimmed milk from a dairy. According to Nocard, the pulmonary tuberculosis of the horse is produced by a bacillus identical with the bacillus of human tuberculosis; while the abdominal form is due to a bacillus resembling that of the cholera of fowls. Dophlide has in one case established a transmission from man. It is very difficult to transmit tuberculosis to the horse by inoculation, though, on the other hand, equine tuberculosis is communicable to fowls.

Anatomical Changes.—The changes are similar to those of tuberculosis of cattle and may become localised in the lungs as well as in the serous membranes; or may become generalised by spreading simultaneously over several organs. We may find in the lungs small miliary tubercles, or large firm nodules which are caseous in their centre, and which may attain the size of a walnut. These nodules may unite into a firm mass infiltrated with yellowish foci, or may form cavities. As in cattle, the lungs may show well-marked symptoms of miliary tuberculosis (multiple embolic foci). Numerous tubercle bacilli are contained in the disintegrated caseous deposits. The bronchial and mediastinal glands are swollen up to the size of a man's fist, are hard to the touch, and have eminences on their surface. Their capsule is thickened and on section they appear grey with interspersed yellow foci. The portions of the pleuræ which cover the tubercles may also become thickened. In one case a tuberculous laryngeal gland was removed from a horse (Nocard).

The serous membranes of the thorax and abdomen often show changes similar to those of bovine tuberculosis. The pleuræ are covered with single or conglomerated grey-yellow, grape-like tubercles, from the size of a poppy seed to that of a hemp seed, and sometimes with larger tubercles which may also become united into thick, nodulose, plate-like layers. Exactly the same changes are found on the perietal and visceral peritoneum, omentum, and mesentery, in the form of nodules varying in size from a millet seed to a walnut, partly isolated, partly strung together in grape-like or plate-like lumps, which may unite together and form tumours that may be as large as a man's fist. The mesenteric glands, like the bronchial glands, suffer change, and form tumours which may be as small as a walnut or

as large as a man's fist, or may assume the appearance of a string of beads that may be as thick as a man's arm, and frequently weigh as much as 44 lbs. Similar changes are sometimes met with in the internal iliac glands. We also find tubercles of various sizes, and sometimes in great numbers, in the liver, in the spleen, which is often enormously enlarged, and in the kidneys, with simultaneous swelling of the portal, splenic and renal lymph glands. Tuberculosis of the bones (ribs and dorsal vertebræ), with disintegration of the bone tissue, formation of caseous foci in the bones, and periosteal proliferations have been observed. It is also stated that tubercular changes occur in the intestine.

Symptoms.—The aspect of equine tuberculosis is not very characteristic. Clinically, we may divide the disease into two principal forms, namely, a pectoral and a gastric. In the former, we usually find only symptoms of "broken wind" or asthma, such as chronic difficulty in breathing, cough, and emaciation, which in some horses progresses so rapidly that they become almost skeletons in the course of from 6 to 12 months. In others, we find foci of dulness in the lungs, bronchial breathing, and râles. In one case, the coughing up of a necrotic, bacilli-laden piece of lung tissue was noticed. A case described by Schindelka reminds one vividly of human phthisis; the symptoms having been bronchitis, rapidly increasing debility, loss of appetite, anæmia, variations in internal temperature, lingering pleuritis, harassing cough, excessive difficulty in breathing, great emaciation and, strange to say, polyuria. Nocard also observed severe polyuria and very irregular increase in temperature, 2° to 3° F. In one instance, he found tubercular deposits in the peritoneum by introducing his hand into the rectum. The diagnostic significance of polyuria and polydipsia in the later stages of equine tuberculosis has lately been brought prominently forward by Knipscheer and Hendrickz.

The second form of tuberculosis is a grave intestinal affection, which is evidently the disease that was formerly described by Strauss, Träger and others under the name of umbilical pyæmia, except when the symptoms were identical with those of pyæmic polyarthritis. The descriptions of the disease correspond exactly with those of tuberculosis of the intestines and mesenteric glands (*Phthisis sive tabes mesenterica*). The foal gets out of health, becomes emaciated, tucked-up in the flanks, and pot-bellied; the

coat becomes harsh and loses its naturally glossy appearance; and the animal suffers from digestive disturbances, such as constipation alternating with diarrhœa, and from slight attacks of colic. It finally becomes debilitated, prostrate, and dies from exhaustion or profuse diarrhœa. On making a *post-mortem* examination, we find enlargement, caseation, and induration of the mesenteric and bronchial glands and catarrhal changes of the gastro-intestinal mucous membrane. Schortmann has described an interesting case of an adult horse suffering from umbilical pyæmia which could be traced to tuberculosis caused by feeding. Finally, in isolated cases, equine tuberculosis takes the form of a breast tumour (Röder, Johne), or an exclusively lymphatic gland tuberculosis of the head and neck (Rabe).

[McFadyean remarks on the frequency with which marked stiffness of the neck is a striking symptom of tuberculosis in the horse; and refers the cause of this stiffness to tuberculous ostitis and periostitis of the implicated cervical vertebræ.]

Differential Diagnosis.—An attempt to distinguish between equine tuberculosis and glanders may be made by means of the inoculation of tuberculin or mallein. We can also form an absolutely correct conclusion on this subject from an exact anatomical and bacteriological examination *post-mortem*. The demonstration of tubercle bacilli by means of the Koch-Ehrlich staining method, or by one of those described on pages 365 *et seq.* of Vol. II., is decisive, as is also the result of the inoculation of guinea-pigs. Csokor points out that characteristic anatomical and histological differences exist between the nodule of glanders and tubercular new growths. He states that the nodules in the lungs in cases of equine tuberculosis are not so well defined as those of glanders; that they possess a distinct inclination to form groups; and that each respective nodule becomes centrally caseated. He also remarks that the nodules (tubercles) in miliary tuberculosis of the lungs possess a fundamental substance which consists of three different kinds of cells, namely, giant cells, epithelioid cells, and round cells. The nodules of glanders, on the contrary, show only one layer of round cells, or connective tissue fibres, around a caseous, disintegrated centre.

Tuberculosis in the Donkey.—Natural tuberculosis is very rare in the donkey. Blanc has hitherto observed but one pronounced case of tuberculosis—in an old donkey used for dissection (miliary tuberculosis of the lungs, inoculation tuberculosis in guinea-pigs). On the other hand, tuberculosis may be communicated artificially to the donkey without difficulty; so that it is not immune to this disease, as has been supposed. Of

11 donkeys subjected to intravenous inoculation, 8 died of tuberculosis; the other 3 were also tuberculous, but recovered with calcification of the nodules (Galtier). Further artificial transmissions have been successfully carried out by Johne, Nocard, Arloing, Galtier, Stockmann and others. Pleindoux has described a case of abdominal tuberculosis in a mule.

4.—Canine Tuberculosis.

Occurrence.—Although tuberculosis is not a rare disease in dogs, it does not occur so frequently in them as in cattle and pigs. During the last 20 years, about 200 cases have been observed: 40 by Cadiot; 40 by Fröhner; 32 by Petit; 28 by Jensen; 13 by Redderoth; 11 by A. Eber, and 10 by von Rätz. Its frequency appears to vary greatly in different countries and towns. Among 70,000 dogs which, from 1886 to 1894, entered the Berlin clinic, 40, that is to say, 0.05 per cent., were affected. Among the 9,000 canine patients at the Alfort clinic during the years 1891 to 1893, 40 were affected, thus giving a percentage of 0.4, which is about 10 times more than in Berlin. Of the dogs slaughtered in recent years in Saxony (300–500 per annum), from 1 to 2 were, on an average, tuberculous (in 1899, 0.2 per cent.; in 1900, 0.16 per cent.). Out of 400 dogs in Dresden, 11 (2.7 per cent.) were tuberculous; in Leipzic, of 1,100 dogs poisoned, 13 (=1.2 per cent.) were found to be tuberculous. The percentage of tuberculosis in dogs was highest in Copenhagen.

Pathogenesis.—The reception of tubercle bacilli in dogs takes place most frequently by means of the lungs, which consequently are more often found (75 per cent. of all cases), on *post-mortem*, to be tubercular than other organs. Infection in them, as in man, is generally transmitted by inhaling tubercular dust in rooms, etc. Less frequently the tubercle bacilli are received through the digestive apparatus, in consequence of swallowing tubercular sputum or tubercular food. Absorption through the skin is rarest of all. It has been amply proved that when the dog becomes tuberculous, the infection has been derived, as a rule, from mankind; especially by inhaling the dust of a room occupied by a consumptive owner, licking up expectorated sputum, and eating food which has been masticated by a phthisical patient. The serious danger to human beings of having tuberculous dogs in their immediate neighbourhood should not be disregarded.

Symptoms.—As in human beings, canine tuberculosis runs

a chronic course usually in the form of pulmonary phthisis. In many cases, no conspicuous external symptom of the disease can be perceived, even for a very long time, on which account owners often remain unconscious of the fact during the life of the animal. The symptoms, however, vary greatly according to the site, extent, and age of the tubercular changes. Usually the disease assumes the aspect of chronic pneumonia, chronic pleuritis, hydrothorax, or chronic bronchial catarrh. The chief symptoms are : cough, dyspnœa, rapidly increasing emaciation, weakness, and a very irregular, atypical fever. Physical examination of the lungs shows dulness, the presence of cavities, rhonchi, suppressed respiratory murmurs, etc. Sputum is seldom observed, because the patient swallows it. Towards the end, there is accompanying diarrhœa and sometimes ascites. By that time the animal is reduced almost to a skeleton. Acute miliary tuberculosis seldom becomes developed; still more seldom does death occur with the symptoms of septicæmia (septicæmic form of tuberculosis, according to Cadiot; mixed infection). Occasionally tuberculosis of the skin has also been observed (ulcers); and unilateral facial paralysis (tuberculosis of the petrous temporal bone).

Anatomical Conditions.—Canine pulmonary tuberculosis assumes many different anatomical aspects. Most frequently we find cavities and caseous foci which usually lead to rupture of the pleural sacs. In other cases, the *lungs* contain large pneumonic or small miliary tubercular foci. We sometimes find chronic indurating broncho-pneumonia, with central softening, or chronic interstitial indurating pneumonia. The lungs frequently adhere to the walls of the ribs and sometimes to the neighbouring lymph glands. Besides this, there may be present excessive compression of the lungs with atelectasis, carnification, œdema of the lungs, emphysema of the lungs, and bronchitis with bronchiectasis.

Tuberculosis of the *pleuræ* appears in various forms. The patient may suffer from either a serous or a sero-fibrinous pleuritis, with considerable discharge, into the thoracic cavity, of fluid, which, in some cases, assumes a hæmorrhagic character; or a dry, granular, callous, adhesive and contracting pleuritis, which leads to adhesions of the lungs. There may also be present a disseminated miliary, pleuro-tuberculosis. Emphysema, and hydrothorax and pneumothorax may be seen as secondary phenomena.

Tuberculosis causes the thoracic *lymph glands* (bronchial glands, anterior and posterior mediastinal glands, and in a few cases the inferior cervical glands) to become more or less enlarged and frequently to adhere to one another, so that they sometimes form sarcomatous conglomerations the size of a man's fist. The tumours show on section a dirty white colour and are infiltrated with foci of softening or tubercles. The mediastinum often becomes thickened and swollen to an unrecognisable extent. In combination with the swollen lymph glands, it sometimes forms between the two lobes of the lung, sarcomatous, lardaceous tumours which are covered with fine proliferations and remind us of those of bovine tuberculosis. These tumours show on section, softened, caseated and calcareous foci.

The diseased condition of the *pericardium* manifests itself partly by a sero-hæmorrhagic, partly by a fibro-granular, adhesive pericarditis, and partly by miliary tuberculosis of the perietal and visceral portions of the pericardium. Hydropericardium is sometimes present.

Tuberculosis of the *peritoneum* is more rare than that of the pleuræ, the changes being almost identical in both. Swollen proliferations, similar to those of the mediastinum, sometimes form on the omentum. We seldom find intestinal tuberculosis (intestinal ulcers with swelling of the mesenteric glands).

Tuberculosis of the *liver*, *spleen*, *testicles*, *kidneys*, etc., is usually miliary.

Statistics.—The respective participation of individual organs in canine tuberculosis is shown by the following figures. According to Cadiot, the lungs were affected in 80 per cent. of all the cases; pleuræ, bronchial glands, mediastinal glands and liver, in 60 per cent.; kidneys, in 45; peritoneum and omentum, in 30; pericardium and mesenteric glands, in 25; spleen and intestinal mucous membrane, in 10; and the heart, in 5 per cent. Exudative pleuritis and ascites were present in 50 per cent.; and exudative pericarditis in 12 per cent. General tuberculosis was met with in more than one-third of all the affected dogs. Jensen states that the lungs were tubercular in 75 per cent. of all the cases. In about half the number, the bronchial glands, mediastinal glands, and serous membranes, especially the pleuræ, were involved. The liver and kidneys were equally often affected. Eber found that the lungs were implicated in 80 per cent. of the cases; the bronchial glands, in 70 per cent.; the pleuræ, in 35; the pericardium, in 10; and general tuberculosis was present in 10 per cent. of the cases. According to Fröhner, the percentage for the lungs, including the pleuræ, was 90; for the liver, 80; for the bronchial and mediastinal glands, 50; for the pericardium, 40; for the kidneys, 25; and for the spleen, and for cases of general

tuberculosis, 10 each. According to Redderoth, tuberculosis of the lymphatic glands of the mesentery occurred in 60 per cent. of the cases without other localisation. Petit found, in 32 cases, pleuritis in 19; pulmonary tuberculosis in 25; pericarditis in 15; and peritonitis in 6.

Diagnosis.—The history of the case in dogs, often leads us to suspect the presence of tuberculosis. We usually learn that the patient has suffered for a considerable time from cough, dyspnœa and increasing emaciation, and that every means of alleviation which had been tried had failed. The phthisical appearance of the owner may lead us to suspect tuberculosis in the dog. Generally, a diagnosis of tuberculosis will be justified, if the dog is suffering from chronic bronchitis, pneumonia or pleuritis, and from great emaciation. An exact diagnosis is, however, possible only by bacteriological examination, inoculation, and, to some extent, by the use of tuberculin. We may find bacteriologically the tubercle bacilli in few numbers in the sputum and in the discharge from the nose (Fröhner and Bang); but can obtain more reliable results by examining the pleuritic exudate procured by puncture of the thorax and by injecting it intraperitoneally into guinea-pigs (Cadiot).

Tuberculin (0.05 gramme (¾ mins.) for large dogs) is in many cases a valuable auxiliary diagnostic agent with dogs. It is a surprising fact that the reaction sometimes takes place as quickly as from 2 to 5 hours after the inoculation. It is, however, unreliable in cases of advanced and general tuberculosis and with feverish animals, and in such cases it is not altogether without danger; for it not unfrequently causes exacerbations, lowering of the internal temperature, and may even set up fatal complications. In some instances, non-tuberculous dogs react to tuberculin.

Therapeutics.—Advanced tuberculosis of the dog is incurable; for which reason and on account of the danger of infection to man, tuberculous dogs should be destroyed without delay. In the early stages of the disease, we may try the effect of strengthening diet, open air, hygiene, and certain medical agents, such as creosote and creolin. In one case, we obtained considerable improvement by continued ½ per cent. inhalations of creolin.

5.—TUBERCULOSIS OF THE CAT.

Tuberculosis is not very rare in cats. As a rule, the source of infection, as in dogs, is obtained from a tuberculous owner. Cats in large towns are especially liable to become affected. In Berlin, out of 100 sick cats, one was tuberculous (Fröhner). Jensen collected 25 cases in Copenhagen. The chief symptoms, as in dogs, are: excessive emaciation, anæmia, debility, dyspnœa, violent coughing, and dulness of the lungs on percussion. On *post-mortem* we find the lungs and the abdominal organs most frequently implicated. Jensen observed that the lungs are very often affected; but the pleuræ, scarcely ever. The mesenteric glands and kidneys suffer in many cases; the intestinal canal and liver rarely. The milk-white colour (from fatty degeneration) of the section surfaces of the larger tubercles is very striking. The lymph glands of the head and neck are frequently attacked. In two cases, the uterus and testicles respectively were tubercular; for which reason Jensen concludes that infection through coition has been absolutely proved in cats. In a few cases, tuberculosis with secondary affection of the axillary glands was found in the subcutaneous tissue, as a result of wounds. One cat suffered from arthritic tuberculosis. Jensen states that cats are affected chiefly through the digestive canal—a fact which we may infer from the frequency of the disease in the mesenteric glands. Sometimes the digestive organs only are affected; although infection through the lungs is not rare. Nocard observed tuberculous arthritis in a cat which had been fed experimentally with milk that contained tubercle bacilli. Bollinger has described two cases of miliary tuberculosis, in which tubercles were present in the pancreas.

6.—TUBERCULOSIS OF SHEEP.

Tuberculosis is rare among sheep. Only 5 cases were found among 340,000 sheep killed in the Berlin abattoir in the course of one year, 1888–1889. Among 130,000 sheep killed in 1894 in Saxony, there was a tuberculous percentage of 0.15. The anatomical changes in ovine tuberculosis closely resemble those of bovine tuberculosis. Several observers suspect that infection is transmitted by tuberculous cattle through cohabitation and milk. Great emaciation, anæmia, and cough are the chief symptoms. In a case described by

Rasmussen, a lean six-year-old sheep, slaughtered in the Copenhagen abattoir, showed a number of defined tubercles, varying in size from a hemp seed to a hazel-nut. These tubercles partly projected out of the surface and were partly imbedded in the tissues. They all consisted of a thick capsule of connective tissue and caseous contents. The bronchial, mediastinal and thoracic glands and the glands under the shoulder blade were considerably swollen, firm, knotty, and studded with a calcareous deposit. Some isolated pedunculated new growths were on the pleuræ, and the liver was partly covered with tubercles and partly infiltrated. The first lumbar vertebra contained a cavity the size of a nut, filled with mortar-like caseous material. Similar processes were found in the two last thoracic vertebræ. Galtier has often produced food-tuberculosis in sheep by repeated administration of materials very rich in bacilli.

7.—Tuberculosis of Goats.

Goats have been considered by many to be refractory to tuberculosis. Acting on this erroneous supposition, goats' milk has been recommended as a substitute for cows' milk, in order to guard the consumer against tuberculosis. In fact, there have been established cure-institutions in which goats' milk was exclusively used. This misconception arose from the fact that formerly tuberculosis was seldom found in goats; because *post-mortem* examinations of these animals were rarely made in abattoirs. Veterinary literature proves, however, that this disease is not at all uncommon in goats. The percentages in Prussia, in the years 1898 and 1899, were 0.5 and 0.4 respectively. In Saxony 0.6 per cent. of the 1,500 goats killed in 1894 were found to be tuberculous; in 1895, 0.4 per cent.; in 1896, 0.3 per cent.; in 1897, 0.37 per cent; in 1898, 0.41 per cent.; in 1899, 0.6 per cent.; in 1900, as much as 2.5 per cent. In Bavaria, in 1898, 8 goats were found to be tuberculous. Of 3,000 goats slaughtered in the abattoirs at Lyons, in 1899, 7 proved to be tuberculous.

In a case reported by Sluys and Korevaar, a goat which had been brought up on cows' milk showed all the signs of pulmonary phthisis, and general tuberculosis, starting from the intestinal tract, was found on *post-mortem*. At the Munich abattoir, Magin observed in a goat that almost all the internal organs, even the spongy tissue of the vertebræ, were infiltrated with tubercles so that the flesh had to be forbidden to be used as food. In spite of the fact that the animal was

well nourished, numerous dirty-white tubercles of a caseous character, varying in size from a millet seed to a pea, were met with in the parenchyma of the lungs, liver, and spleen, and in the pleuræ and peritoneum. The mediastinal glands were also swollen and infiltrated with tubercles. Edgar found in a five-year-old goat the entire lung infiltrated with small, greyish-yellow nodules. Eichhorn inoculated 28 goats out of a herd of goats in which a case of tuberculosis had occurred, with from 0·02 to 0·05 grammes ($\frac{1}{3}$–$\frac{3}{4}$ mins.) each of tuberculin; the result being that, with a single exception, all the animals showed an increase of temperature of from 1° to 5° F.; and eighteen of them, a rise of over 2° F. Three of these animals which were slaughtered, proved to be tuberculous. In all probability, the majority of the remainder was affected. Möbius found tuberculosis of the lungs, liver and testicles in a he-goat.

Tuberculosis in Wild Mammalia.

Of the wild animals kept in zoological gardens, many are affected with tuberculosis; but the great majority are *monkeys*. As Nocard has shown, the tuberculosis of monkeys is conveyed by the digestive organs. According to his researches, also, monkeys are as susceptible to infection with bovine tuberculosis as other mammalia; while in comparison with the human bacillus, the bovine bacillus is much more virulent and kills more rapidly. According to De Haan, the lungs are also, in monkeys as in man, the favourite seat of tubercular disease. Tuberculosis has also been observed in menageries in lions, tigers, panthers, giraffes, etc.

8.—Tuberculosis of Birds.

Occurrence.—Tuberculosis is one of the most common diseases of birds. Amongst 1,100 fowls dissected at the Leipsic veterinary clinic, 106, namely, 10 per cent., were found to be tuberculous. Among 700 parrots treated, from 1886 to 1894, in the Berlin clinic, 170 (25 per cent.) were similarly affected. This disease attacks all kinds of birds (hens, pigeons, pheasants, peacocks, parrots, etc.), and not unfrequently occurs as an enzoötic. In poultry, tuberculosis is generally located in the intestines, which, consequently, is the usual entrance of infection. The transmission usually takes place by means of the fæces of affected fowls, which fæces contain great numbers of bacilli. The infection is sometimes derived from tuberculous human beings by the eating of tubercular sputum and of food which had been previously masticated. It

also appears that the disease is transmitted by the milk and flesh of tuberculous cattle. The heredity of tuberculosis, which is credited by poultry breeders, has been proved by the experimental investigations of Maffucci. On the pathogenesis of the tuberculosis of parrots, see below.

Recently doubts have been expressed about the identity of the tuberculosis of birds with that of mammals, including man. Koch considers that the bacilli of avian tuberculosis differs from true tubercle bacilli, although it is closely related to them. He leaves open the question of their having the same pathogenic effect on man. Rivolta, Maffucci, Baumgarten, Strauss, and Gamaleïa have also pronounced in favour of making a distinction between the two diseases and between the two bacilli; because human tuberculosis can be easily transmitted by inoculation to guinea-pigs, which are almost immune from the tuberculosis of birds. In contrast to human tuberculosis, the tuberculosis of fowl can be easily transmitted to fowl and rabbits. The more recent investigations of Cadiot, Gilbert, Roger, Fischl, Courmont, and others, tend to show that these two forms of tuberculosis are produced by the same species of bacilli, although by different varieties; the differences between them having been produced by changes in their respective modes of nutrition. Nocard has recently succeeded, by breeding the bacilli of human tuberculosis and of bovine tuberculosis in collodion bulbs within the abdominal cavity of fowls, in conferring upon them the same biological and pathological properties which characterise the bacillus of the tuberculosis of birds. The latter is, nevertheless, according to Nocard, more widely removed from the bacillus of human tuberculosis, than that of the bovine form of the disease. The tuberculosis of parrots appears, on the other hand, to be identical with tuberculosis of mammals (see next page).

Anatomical Conditions.—In poultry, the anatomical changes are chiefly found in the liver spleen, and intestines. The liver is infiltrated, especially on its surface, by tubercles, varying in size from a millet seed to a pea, or even a walnut. These tubercles frequently become massed together, and show, according to their age, a white, grey, or yellow colour. In the centre they are caseous or calcareous, and contain great numbers of tubercle bacilli. Similar changes are found in the spleen. On the intestinal mucous membrane we may see miliary tubercles about the size of a pea, which subsequently ulcerate.

Tubercles also form on the peritoneum. mesentery, kidneys, ovaries, etc. Tuberculosis of the lungs, guttural pouches, heart, and pericardium is rare. The disease frequently invades the lymph glands and articulations, especially those of the wings and feet, in the neighbourhood of which large swellings and abscesses with caseous contents develop, thus causing tubercular arthritis and periarthritis. Tubercles and ulcers also form in the bones, skin, and subcutaneous cellular tissue.

Symptoms.—The symptoms of tuberculosis of birds are not very characteristic. The sufferers are feeble and become gradually emaciated, often almost to skeletons. The comb and wattles are pale and dried up. The mucous membranes of the eyes and mouth are pale, and the birds suffer from marked chronic disturbances of digestion, such as loss of appetite, vomiting, and diarrhœa. Towards the end, great debility and paralysis supervene. The diagnosis becomes somewhat easier when these symptoms are associated with local tuberculosis in the articulations, bones, and skin, in the form of swelling of the joints and bones, and of tumours and unhealthy ulcers in the skin. The disease usually runs a slow course.

Tuberculosis of Parrots.

This form differs in some points from that of domestic fowl. The infection usually takes place through the respiratory apparatus from the birds inhaling the bacilli of human sputum along with dust deposited in dwelling-rooms. Next comes infection through the digestive apparatus, by eating food which had been masticated and then rejected by the owner of the parrot. The skin, especially that of the head, and the mucous membrane of the eyes also form, at times, the port of entrance. Tuberculosis of the skin is characterised by new growths of horny skin, which frequently attain a considerable size. They can be easily broken off, and contain numerous tubercle bacilli in their basal granulation tissue. Tuberculosis of the tongue and conjunctiva is often met with. According to Eberlein, the skin was infected in 52, the tongue in 16, and the bones and articulations in 12 per cent. of all the cases. The tubercles of the skin vary in size from the head of a pin to a hen's egg; are spherical or oval and semi-solid; and have a horny, scaly surface. From a diagnostic point of view, it is important to note that the tuberculosis of parrots is distinguished by the presence of an enormous number of bacilli. Eberlein found numerous bacilli, on cutting and making punctures into the new growths, and by examining the fæces, nasal discharge, and saliva. The treatment of tubercular new growths belongs to the domain of surgery. Relapses are common.

Tuberculosis of Tortoises.

According to Friedmann, the tortoise tubercle bacillus, a species distinct from the tubercle bacillus of man, has an enormous distribution in the bodies of all cold-blooded animals. Tortoises, ringed snakes, lizards, blind-worms and frogs sicken very rapidly with miliary tuberculosis. On the other hand, most warm-blooded animals are immune (birds, dogs, rats, white mice, rabbits). Guinea-pigs only succumb, in from 4 to 8 days, after the intraperitoneal injection of very large doses of tortoise tubercle bacilli (caseating, fibrinous peritonitis with formation of tubercles).

Pseudo-Tuberculosis.

This name is given to some diseases which closely resemble tuberculosis, anatomically and clinically, but are not caused by the bacillus of tuberculosis. These diseases are of diagnostic importance from the fact that they occur in cattle, and in animals which are used for inoculation experiments, in both of which cases they may become confused with true tuberculosis. A correct conclusion can be arrived at only by bacteriological means.

1. Kitt has described in *cattle* a bacillar, caseous, broncho-pneumonia which could not be clinically distinguished from tuberculosis. On bacteriological examination he found bacilli of from 1 to 1½ μ long, of about the thickness of the bacilli of erysipelas and capable of being stained with Gram's solution; but no tubercle bacilli. A similar disease has been described as "bovine glanders," by the French veterinary surgeon Nocard. Courmont observed a peculiar bacillus in tubercular-like pleural nodules of cattle. Stöhr noticed in sucking calves a caseous non-tubercular pneumonia with a singular bacillus. We may draw attention to the pseudo-tubercular pneumonic foci which are met with in diseases caused by pulmonary worms (strongyli) in cattle and sheep, such as parasitic tuberculosis due to nematodes or "nematode tuberculosis." Hink has observed in the lungs of a large ruminant, tubercle-like formations caused by "aspergillus fumigatus." Preisz, Guinard and Tusski found small short bacteria with rounded ends in the tubercle-like calcareous nodules of the kidneys of a *sheep*. In Argentine cattle, there are often found tubercle-like nodules in the small intestine, which are due to sclerostomum larvæ. Preusze has described a pseudo-tuberculosis of the lymphatic glands (caseous lymphadenitis) in sheep; a similar disease seems to appear frequently in Australian sheep (Cherry and Bull). Also in various districts of North America, the caseous disease of the lymphatic glands of sheep occurs pretty frequently; the prescapular, inguinal, popliteal, mediastinal and tracheal glands increase to about four times their normal size; a considerable failure of nutrition is, however, not present during the process of encapsuling of the caseous masses (Norgaard and Mohler). Also in New Zealand (Gilruth), and in Italy (Dessi and Tosi), this lymphadenitis caseosa has been observed. The short bacillus, 0.5–1 μ in length, and 0.3 μ in width, sometimes also produces necrosis in the lungs (subpleural nodules). Pseudo-tuberculosis in the lungs of a *goat*, produced by a pathogenic species of streptothrix, has been observed by Silberschmidt. De Jong has published a report on pseudo-tuberculosis in sheep and goats, produced by *strongylus rufescens.*

2. In *guinea-pigs* and *rabbits* we meet with various processes which resemble tuberculosis. Eberth, Dor, Parietti, Zagari, Chantemesse, Charrin and Roger have described affections closely resembling either tuberculosis or glanders in these two very important experimental animals. These simulating diseases could be distinguished from the true ones only by their respective bacteriological differences. Pfeiffer described in detail a specific and pathogenic *bacillus pseudo-tuberculosis*, which he considers to be the cause of the so-called pseudo-tuberculosis of guinea-pigs and rabbits. This bacillus can be stained by Löffler's solution but not by that of Gram. It has many points of resemblance to the bacillus of glanders, but differs essentially from that of tuberculosis. Inoculations of it produce caseous nodules in the liver and spleen, varying in size from a pin's head to a pea, and caseous foci in the lungs resembling the growths of glanders. The fact that the abdominal organs participate in the disease to a far greater extent than the lungs, serves to macroscopically distinguish pseudo-tuberculosis from true tuberculosis. The pseudo-tubercular nodules contain no giant cells, and only a few epithelioid cells. The inoculated animals die quicker than those inoculated with true tuberculosis, on which account the nodules never undergo dry caseation, but show in their centre a pus-like fluid, as in glanders. On pseudo-tubercle bacilli occurring in cow's milk, which are pathogenic to guinea-pigs, see Vol. II., p. 367.

3. Ebstein and Nicolaïer have found in the kidneys and lungs of *dogs* and *cats* a zooparasitic tuberculosis, which appears in the form of nodules, and is caused by cylindrically-shaped worms. According to Laulanié, similar nodules are produced in the lungs by strongylus vasorum. Potain has described in pigeons, tubercle-like formations caused by aspergillus, and Cornil has reported the presence, in an antelope, of similar affections caused by an oval bacterium.

4. Deleïdi found tubercle-like nodules, which were apparently produced by *strongylus rufescens*, in the livers of *pigs*. Terni has observed similar nodules in the lungs, produced by special bacilli.

ACTINOMYCOSIS.

Actinomycosis in Pigs, Horses and Sheep—Actinomyces Musculorum suis—Actinomycosis of Man.

Etiology.—Actinomycosis is due to ray fungi (actinomyces), which belong to the pleomorphous bacteria and to the cladothrix group of fission algæ, or fission fungi (Boström). According to recent investigations, they appear to belong to the family of streptothrix and to occur in different varieties. The characteristic feature of these fungi, as compared to other bacteria, is that they grow, not as single individuals, but as regularly built-up, combined structures. These colonies or granules measure $\frac{1}{5}$ to $\frac{1}{2}$ mm. ($\frac{1}{125}$ - $\frac{1}{50}$ inch) in diameter, and may be seen

by the naked eye as spherical bodies which are about the size of grains of sand. According to their age, they are grey and gelatinous, and resemble small lumps of mucus; or they are greyish-white, opaque, and sometimes yellow, brown, or even green. We can distinguish in the radially arranged colonies, two layers, namely, a peripheral layer of clubs, and a central layer of threads. The former consists of pear-shaped bodies, the so-called clubs, in the axis of which runs a thread of mycelium; the contents breaking up into spore-like granules. When these clubs open out at their apex, after the manner of a bud, secondary clubs become developed, in the form of finger-like or hand-like processes. As demonstrated by Boström, these clubs are not organs of fructification (gonodia), but are forms of gelatinous degeneration. The central thread layer consists of an entangled plexus of filaments formed by rods, which are of various lengths and are motile in water, and by long spiral threads. Moreover, we find a granular substance consisting of round cocci-like structures (spores) which have sprung from the filaments. The true organs of fertilisation are found in the interior of the actinomyces-granules, in the germ stratum of the filamentous layer. The growth of new colonies takes place by the separation of the filaments or spores of the old granules. It is probable that the germs of the fungus in the form of rods or filaments are carried away by the leucocytes (phagocytosis and chemiotaxis). The tissues lying in the neighbourhood of the tufts die and become surrounded by an outer zone of leucocytes, and later on by granulation tissue. The gelatinous degeneration of the nodules (formation of clubs) is accelerated by increase of granulation tissue. The tufts frequently become calcareous.

According to recent researches the actinomycosis bovis and hominis are not identical; there are different varieties of actinomycosis fungi met with both in cattle and in man.

Bacteriology.—Pure cultures of actinomyces on dried blood serum, which are best prepared by previously rubbing down the granules, form at first, according to Boström, a thin gelatinous layer, consisting of fine transparent filaments. The layer grows and looks as if it were sprinkled over with chalk in consequence of the accumulation of little dots containing numerous cocci and filaments. After a fortnight, these white dots become pale yellow, pure red, or brick red in the centre, and confluent; whilst on the periphery a transparent dirty-white stratum forms. Old cultures become puckered and hard. The fungus grows in a similar way on agar-agar, glycerine agar, and gelatine. On potatoes it forms grey, yellowish, and finally white granules. The growth is not stopped by the exclusion of air (facultative anærobic bacteria). Experiments with

cultures in animals have been mostly negative. Only Wolff and Israël have succeeded in producing actinomycosis by inoculation.

The actinomycosis fungus, according to recent researches, occurs in different varieties, both in animals and man. Gasperini was the first to observe that the actinomycosis of cattle was produced by several varieties of fungi (sulfureus, albus, luteo-roseus). Lignières and Spitz state that there occurs in the Argentine, in addition to the true actinomycosis, an actinobacillosis, the bacilli of which do not stain by Gram's method. It is also clinically distinguished by the usual involvement of the lymphatic glands (which is rarely found to occur in the true form), and the greyish-white colour of the masses (in contrast to the yellow). Jelenewski professes to have found in the labial actinomycosis of cattle, a specific *actinomyces labiatus bovis*. Levy, Bruns and others have further announced that, in human beings, different varieties are found (aërobic, anaërobic); and, further, that the *actinomyces bovis* and *hominis* are not identical. According to Wolff and Israël, the view of the existence of various actinomyces is not justified; the variations in size, etc., being accounted for by the differences of the age of the parasite and of the duration of the disease.

Recent researches have shown that the formation of knobs is not characteristic of the actinomycosis fungus. According to Lubarsch, the club-shape is not a degenerative malformation, but one due to obstruction, and is widely distributed among bacteria. It is found in the bacilli of tuberculosis, in the fungi of pseudo-tuberculosis, and in streptothrix. Schulze found, likewise, a club formation after inoculation with cultures of tubercle bacilli; and believes that it is the result of the overpowering of the tubercle bacilli by the energy of the surrounding tissues. Babes has even expressed the opinion that actinomycosis and tuberculosis are but different forms of one and the same disease.

Pathogenesis.—Actinomyces, which are found originally on plants, seem capable of entering the body in various ways, although the digestive canal is usually their first point of attack. Brazzola considers that they vegetate chiefly on *hordeum murinum*. He discovered quantities of them between the vegetable fibres of broken particles of barley which were imbedded in the gums. Johne, Piana, Boström, and others have found this fungus abundantly among the awns of corn which were in the tonsils of pigs and in the tongues of cattle. It is generally supposed that transmission takes place by the fungus settling in small wounds on the buccal membrane, in the ducts of the glands, and perhaps in the alveoli of diseased teeth, or during the shedding of the milk teeth; and that it grows and develops from these sites. It seems, especially with cattle, that the awns of grain, covered with this fungus, penetrate between the teeth and into the gums and tongue. If they have once effected a lodgment, they can be removed only with difficulty on account

of the peculiar position of the hair on the beard, just as we see with spikes of rye which have become stuck on the sleeve of a coat. A favourite site for actinomycosis of the tongue is a point on the upper surface of the tongue midway between the dorsum and the tip. Hentschel and Falk point out that about 9 per cent. of affected cattle showed on that spot, lesions of epithelium which served as starting points for the infection. Boström believes that the ray fungus develops exclusively on grain, particularly on the awns of barley; and that actinomycosis is caused only by the penetration of such infected parts of plants. We find in dry grain, symmetrically-arranged air spaces, which have fissured openings in which the fungus may remain dormant for more than a year in a dry condition. After that, when it is transferred with the awn to animal tissue, it begins to grow luxuriantly. It appears, as a rule, that cattle become infected only when they are fed on dry food (Imminger and Claus). The immigration may, however, begin in the lungs by fungi contained in the inhaled air; in wounds of the skin; and by the openings of the ducts of the teats. Gooch observed that in a large herd of cattle which had been treated by setons, the majority of the wounds showed actinomycosis. A general infection over the whole body by means of the blood, as in tuberculosis, seems possible, although it could rarely occur. With regard to the rapidity of growth of the actinomycomata, Andersen observed that tumours of greater or less size developed in 17 cows out of 20 which were out on grass during the 5 months from July to December.

Some observations suggest the idea that *swampy districts* are favourable to the development of actinomycosis. Bang and Jensen remarked on the epizoötic distribution of the disease among the cattle of a seaside district of Denmark, in which place the sea had receded from a portion of a bay and left a lake on the reclaimed land, which was exposed to inundations. Cattle which had been fed on barley grown on the reclaimed ground of this inundated district acquired the disease. Imminger, Preusse, Claus, Roger, Davaine, and others, have made similar observations on the influence of swampy and inundated pastures.

Actinomycosis is not a contagious disease. Attempts to transmit it to other animals have been made by various persons, but always without result (Rivolta, Bollinger, Siedamgrotzky, Perroncito, Johne, Ullmann, Bodemer, and Boström). Cattle, calves, goats, sheep, pigs, dogs, cats, rabbits, and guinea-pigs have remained healthy after inoculation. Only in a few cases, re-

action to inoculation was displayed by the formation of inflammatory demarcation granulations. The asserted positive results obtained by Ponfick, Israël, Rotter, and Hanau are, according to Boström, simply residual and encapsuled inoculation nodules. The ray fungus appears to have a pathogenic effect only in the stage of development connected with the awns of grain, and to lose its power of transmission as soon as it has entered the animal body, on account of undergoing some form of involution (calcification, etc.). The negative result of the inoculation experiments is of great importance for elucidating the question of the transmissibility of actinomycosis from one animal to another, or from one of the lower animals to man. The foregoing considerations tend to prove that infection cannot take place in this manner. More especially, also, do the experiences of meat-inspection disprove the occurrence of a direct communication (see next page).

Occurrence.—Actinomycosis has been observed in cattle, pigs, horses, sheep, and man. The ray fungus was first seen about 1860 by Perroncito and Rivolta, and by Hahn in 1870. Bollinger furnished the first exact description of the disease in 1877. Harz was the first to examine the fungus botanically and to give it the name actinomycosis (ray fungus).

Actinomycosis is generally sporadic, though sometimes enzoötic. Preusse has described an epizoötic outbreak of this disease in West Prussia, by which 20 per cent. of all the cattle became infected. Imminger states that the disease is particularly rife in the Upper Palatinate and neighbouring parts of Upper Franconia. He is accustomed to treat yearly more than 100 cases of bovine actinomycosis in the Bavarian Upper Palatinate. Claus has collected 105 cases. Actinomycosis appears to be very common in Russia. Of all the cattle slaughtered in Moscow, from 2·5 to 5·5 per cent. are found to be infected with it (Oskolkow). Barret states that Canada shows the comparatively high percentage of 2 ; Salmon puts the percentage for the remainder of North America down at 0·2. The disease is somewhat rare in France (Cadiot). The percentage in La Vilette was 0·7 (Nocard). Peletti has seen hundreds of cases of actinomycosis in Italy. His experience of the disease impressed upon him the fact that it is especially liable to occur after an epizoötic outbreak of aphthæ, on which account he connects the presence of ulcers in the buccal membrane with the pathogenesis of actinomycosis. Neuwirth has made a similar observation.

In Denmark the outer soft parts of the face and the neighbouring cervical region are most frequently attacked (Bang). In England the usual seat of the disease is the tongue; and in Russia, the lips. In Sweden, according to Vennerholm, 3,560 cases of actinomycosis occurred in the years 1890 to 1892, of which 626 ended fatally. In Hungary, actinomycosis is widely distributed in some localities; Breuer found it, in winter, in 33 per cent. of slaughtered cattle; in 16 per cent. during summer. In Switzerland, Ehrhardt ascertained the existence of a percentage of 0.1 to 0.6 of the cattle treated in Zürich during the years 1888 to 1895.

The *Statistics of the Abattoirs* give the following figures. In Berlin there were 21 cases from 1885 to 1886 among 100,000 cattle (1 in 5,000); and 2 out of 300,000 pigs (1 in 150,000). In Augsburg, 8 in 23,000 cattle (1 in 3,000). In Bremen, 2 among 8,500 cattle (1 in 4,250), and 3 out of 25,000 pigs (1 in 8,000). In Stuttgart, 12 among 12,000 cattle (1 in 1,000); and in Hanover, 1 in 10,000 cattle. Ivanow found 2,000 cases of actinomycosis during 2 years in the abattoir of Moscow, in the slaughter-house of which Mari observed 540 infected carcases out of 150,000 (1 in 3,000) cattle that were killed. At Warsaw there were 70 cases out of 350,000 beasts (1 in 5,000).

Anatomical Conditions.—Boström states that actinomycosis is a specific inflammation which occurs in the three following degrees in our domestic animals: (1) A degenerative granular inflammation (tongue); (2) a progressive purulent granular inflammation (cold abscesses); (3) a fungous actinomycoma (pharynx and skin). First of all a granulation tumour develops in the neighbourhood of the actinomycotic granules, in consequence of inflammatory reaction. This tumour leads to the formation of tubercle-like nodules and large rounded or lobulated tumours, which are termed actinomycomata. These actinomycomata are sometimes very soft, of a sarcomatous consistence, and of an orange colour. At other times they are rather hard and firm to the touch, of the consistence of a fibroma, of a whitish-grey colour, and sometimes spongy. They consist of a connective tissue stroma in which numerous nodules the size of a millet seed up to that of a pea are interspersed, and shine through the surface. These nodules possess the microscopic structure of granulation tumours and contain the sulphur-yellow actinomycosis granules, which are about the size of a grain of sand. They may form larger nodules by becoming massed together. Purulent disintegration of the actinomycomata gives rise to "cold abscesses," which are surrounded by very soft

granulation tissue, and contain the small yellow tufts of the fungus. The actinomycotic masses are usually surrounded by a strong wall of connective tissue.

Actinomycosis of cattle usually appears in the upper or lower *maxillary bones*, where it generally produces very large tumours in their soft tissues and osseous tissue. These tumours were formerly known under the name of "wens," spina ventosa, sarcoma of the jaw, cancer, etc. Actinomycosis of the jaw commences with flat granulations on the gums and mucous membrane in the neighbourhood of the teeth, and spreads to the periosteum, and finally to the medullary tissue of the bones, where it soon gives rise to an osseous tumour. This swelling is the product of an ossifying periostitis, and of a rarefying granular osteitis, which gives rise to the spongy cavities, or lacunæ that are highly characteristic of macerated bone, and are due to atrophy of the osseous trabeculæ. On the other hand, the simultaneous periostitis is followed by an increase of the circumference of the bone with a peripheral osseous deposit. From the maxillary bone, the actinomycotic granulation substance may advance, either to the skin or to the oral cavity, in the direction of the molar teeth, which become displaced, or finally get affected by the morbid process in the same manner as the bone.

In cases of actinomycosis, the disease very frequently attacks the *tongue*, in which it takes essentially the form of an indurating glossitis (actinomycotic sclerosis or macroglossia), for which reason it was formerly called "wooden tongue" (*Holzzunge*, and *langue de bois*). We notice, first of all, underneath and on the sides of the mucous membrane of the tongue, circular, somewhat raised brown spots, through which shine very minute yellow nodules. Later on, we find similar spots, varying in size from a grain of millet to a pea, in and under the mucous membrane and in the intramuscular connective tissue. These actinomycotic nodules are round, hard to the touch, fibrous, and show a purulent, caseous, chalky or mortar-like substance in their centre. In the neighbourhood of these nodules there is a considerable proliferation of connective tissue, which leads to atrophy of the muscle fibres, or to induration of the organ ("wooden tongue"). On cutting through the tissues of the greatly enlarged tongue, we find it lardaceous, hard, and even gritty. The cicatricial shrinking of the proliferated connective tissue eventually causes contractions, erosions from friction, and various deformities of the tongue. The lymph glands of the tongue are studded with nodules and abscesses.

As a rule, the actinomycotic proliferations in the *pharynx* take the form of soft polypoid or fungoid nodules or lumps which have a short peduncle and smooth surface, and vary in size from a pea to a goose's egg. These actinomycomata may give rise to difficulty in swallowing and attacks of dyspnœa. Similar nodules which sometimes have a broad base, and which vary in number, size, and consistence, may form in the *œsophagus*, *reticulum*, *larynx*, and *trachea*. Actinomycotic processes are of rarer occurrence in the *omasum*, *abomasum*, *intestines*, *peritoneum*, *omentum*, and *mesenteric glands*.

Actinomycomata of the *skin* and *subcutis* are found chiefly on the head and neck. They usually form elastic, firm nodules, the size of a hazel-nut up to that of a man's fist, or may be even larger. Sometimes these nodules are pedunculated; at other times they are attached to the skin by means of a broad base. They may present the form of a soft, granular, fungoid proliferation of the colour of red meat, and are then covered with brown crusts, or with a purulent secretion. At other times, yellow nodules, the size of a pin's head, shine through the surface of these proliferations, in the neighbourhood of which the skin becomes thickened and indurated. Cutaneous actinomycomata may attain a very large size. Actinomycosis of the skin may be primary or secondary. In the latter case, it takes its starting point from deep-lying primary foci, in, for instance, the jaw, udder, or glands. Tumours and ulcerous erosions form on the mucous membranes, chiefly of the mouth and nostrils.

In many cases the *lymph glands* in the neighbourhood of the larynx and the pharynx are not infrequently affected secondarily in actinomycosis; as the infective process extends from the buccal cavity, the larynx, the throat, etc., to the neighbouring lymphatic glands. Actinomycoma is most frequently found in the sub-parotidean (upper cervical) glands. This condition was formerly known as "parotid gland lymphoma"; "glandular tubercle"; "scrofula nodules," etc. They form firm, round, oval, or even elongated nodules, the size of a walnut up to that of a man's fist, in which typical masses of threads are to be found on microscopic examination. The sub-maxillary, parotid, and sublingual glands sometimes become affected.

Actinomycosis of the *lungs* occurs in two forms: First, that of disseminated, firm, whitey-yellow nodules which become calcareous in their centre and vary in size from a millet seed to a pea (miliary actinomycosis); second, that of larger purulent foci of softening (cavities), which may attain the size of a man's

fist, and which are filled with a grey, muco-purulent fluid (lobular actinomycotic inhalation-pneumonia). The actinomycomata of the lungs may spread to the pleuræ, and even reach the surface of the body by penetrating through the ribs or intercostal muscles. The bronchial glands and the mucous membrane of the air passages of the head may become affected.

Actinomycotic processes may appear on the *udder*, in the form of soft suppurating masses, varying in size from a bean to a goose egg, with surrounding fibrous hardening (mastitis actinomycotica purulenta fibrosa), rarely in the form of disseminated actinomycosis of the udder, in the *spermatic cord* of castrated animals, *brain* and its membranes, *spleen*, *liver* (in one case described by Willach the weight of the affected liver amounted to over 116 lbs.), *muscles*, *bones* (breast-bone, dorsal and cervical vertebræ, ribs, tibia, metatarsus), *kidneys*, *diaphragm*, *peritoneum*, *inguinal glands*, *bladder*, *vagina*, *uterus*, *testicles*, *ear*, etc. Actinomycosis, like tuberculosis, may become general.

Statistics.—The maxillary bones, tongue, pharynx, parotid glands and skin are the most frequent seats of the disease; although there are exceptions to this rule. Among the 105 cases collected by Claus, the maxillary bones (the lower jaw in the majority of such cases) were affected in 51 per cent.; the tongue, in 29; the pharynx, in 7; the larynx and trachea, in 6; and the lungs, abdominal viscera and bones of the head, only in a few instances. According to Imminger, the head and neck are implicated in from 85 to 90 per cent. of the cases, and the tongue in only 4 to 8 per cent. Rasmussen found the jaw affected in 14 out of 15 cases in oxen. Among 201 diseased cattle observed by Kuritzin, there were only 3 maxillary cases; the tongue being almost exclusively attacked. Actinomycosis of the tongue appears to be particularly rare in France, in which country out of 130,000 cattle which showed 0.7 per cent. of actinomycosis, that organ suffered only in one instance. Oskolkow states that in Moscow the lips were involved in 50 per cent. of the cases. On the other hand, among 55,000 cases of actinomycosis which occurred there during the years 1894 to 1900, the lungs were affected in 500 only (0.9 per cent.). A primary miliary form of actinomycosis in cattle has been observed in the abattoirs of Lyons (numerous hard white, suppurating nodules of the size of a millet or hemp seed, with numerous granulations of the size of a pea in the place of the retropharyngeal glands). Hutyra states that the actinomycosis of a Hungarian county, which was specially infected with this disease in 1898, was located as follows: neighbourhood of throat, 50 per cent; cheeks, 30 per cent.; cavity of mouth, 15 per cent.; skin, 5 per cent. According to the collective statistics of Jelenewski, of the total number of the cattle slaughtered during the five years, 1896 to 1901, in Berlin, 0.3 per cent.; in Moscow, 3.3 per cent.; in Kiew, 0.7 per cent.; in Warsaw, 0.6 per cent.; and in Jelisawetgrad, 3.3 per cent. were affected with actino-

mycosis. Of the entire number, 38 per cent. were cases of actinomycosis of the lips in Moscow; in Tiflis, 86 per cent.; and in Jelisawetgrad, 82 per cent. were of the same. Mari gives the following figures from 541 cases of bovine actinomycosis which he collected:—Skin, 271; submaxillary glands, 177; bones of the head, 117; lymph glands under base of skull, 51; upper bronchial glands, 38; lungs, 29; inferior bronchial glands and tongue, 5; pharynx and bronchial glands, 4; peritoneum, 3; pelvis, 2; pleuræ, inguinal glands, wall of the thoracic cavity, wall of abdominal cavity, mediastinal glands, and liver, 1 each only.

Actinomycosis in Pigs, Horses, Sheep and Dogs.—In *pigs* actinomycosis appears in the lower jaw, larynx, lungs, wounds caused by the castration of males and females, mammæ, flesh, and bones. When the bones are implicated, they become permeated with purulent cavities and sinuses, the contents of which show numerous yellow granules. Korsak frequently found in the tonsils of pigs the awns of barley which were covered with proliferations of actinomyces. Actinomycosis of the udder appears in numerous pustules from the size of a pea to that of a walnut—that is abscesses which are embedded in firm fibrous tissue—and at times break through the skin. According to Hamoir, actinomycosis of the udder specially affects the space between the teats. Carl has described a diffused actinomycosis of the stomach and intestinal canal. The presence of general actinomycosis has been seen in pigs.

In *horses*, cases have been observed of actinomycosis of the bones, tongue, abdominal wall, laryngeal lymphatic glands (may be mistaken for glanders), spermatic cord and submaxillary glands. In three horses which reacted typically to mallein, numerous actinomycotic nodules were found in the lungs, liver, and spleen; varying in size from that of a millet seed to that of a pea, and partly calcified (Bruschettini). In a case described by Hartl, the whole larynx was filled with a solid, flat tumour, which reached to the cheeks, and from which strings of lymphatics extended to the lips, like strings of pearls (confusion with glanders).

A few cases of actinomycosis of the lungs and muscles have been described in *sheep*. Berg has observed three cases in sheep (tongue, lips).

In *dogs*, isolated cases of actinomycosis have been observed, and also in *cats* by Hartl, Gohn and Torrance (tumours on the thorax, in the thoracic cavity, and in the buccal cavity in dogs; purulent peritonitis in cats).

Symptoms.—Although actinomycosis is of greater interest to surgery and pathological anatomy than to internal medicine, it sometimes presents symptoms which belong to the domain of special pathology. Thus, in actinomycosis of the tongue, prehension of food, and especially mastication, are impeded and even rendered almost impossible; the tongue is swollen and painful to the touch; and copious salivation is frequently present. Difficulty in swallowing arises from implication of the pharynx; dyspnœa, from new growths in the larynx; and chronic tympanites, from actinomycosis of the mediastinal lymph glands.

In cases of actinomycosis of the outer surface of the neck, we find the parotid region swollen and covered with tumours of varying size. Actinomycosis of the cervical vertebræ may cause spinal paralysis as a consequence of atrophy of the spinal medulla from continued pressure. Actinomycosis of the lungs may also present the appearance of a chronic affection of the lungs (phthisis). The course of the disease is always of long duration. Cases of spontaneous recovery may take place, as in tuberculosis, in consequence of the growth becoming encysted and calcareous. Recent experiences with iodine have rendered prognosis more favourable. Actinomycomata of the skin, glands, and maxillæ can be treated surgically.

Differential Diagnosis.—The diseases which are most easily mistaken for actinomycosis are tuberculosis (tuberculosis of the lungs and lymph glands), bovine pleuro-pneumonia, and foot and mouth disease. It may be mistaken for simple glossitis showing new growths in the pharynx (polypi, fibromata, and sarcomata), with glanders (horses), sarcomata of the jaw, lymphomata, cysticercus mediocannelatus in the tongue, parotitis, cellulitis, etc. On this account, the diagnosis of actinomycosis during the life of the patient is by no means easy. Often the disease can be recognised only by a microscopical examination after slaughter. For diagnosis *intra vitam*, it is of importance to extirpate or to incise nodules or abscesses in or under the skin, and to submit them to an exact microscopical examination. The actinomycotic erosions which occur on the mucous membrane of the mouth differ from aphthous ulcers, by reason of their base being hard and leathery. In all cases, the diagnosis can be confirmed only by microscopical proof of the presence of the actinomyces tufts.

Simple microscopical examination with the addition of one drop of concentrated liquor potassæ suffices to demonstrate the presence of the ray fungus. The staining of the fungi gives a great deal of trouble and is not absolutely necessary. Among different colouring materials which have been used, we may mention a double staining with orseille and gentian violet, orcein, picro-carmine, hæmatoxylin, eosine and a mixture of aniline dyes. The cover glass preparations are made in the ordinary way. A small quantity of the pus or other material which is to be examined, is spread in as thin a film as possible on a cover glass. It is dried in the air and then passed, with the film side upwards, three times through a spirit flame, by which it must not

be heated too highly. The cover glass preparation is then placed for two or three minutes in a solution of picrocarmine, or other colouring agent, after which it is cautiously rinsed in water or alcohol and examined in water, glycerine, or Canada balsam. If the ray fungi be present, they will show a bright yellow colour and the tissue will manifest a red stain.

Therapeutics.—Formerly the treatment of actinomycosis was exclusively surgical, as for instance, by extirpation, corrosion with arsenic, chromic acid, actual cautery, incision, parenchymatous injection, and painting, which purely operative methods, especially that of extirpation with the knife, are even now applied, as the surest, to actinomycomata that can be reached externally. The deeper lying tumours, especially actinomycomata of the pharynx and the larynx, cannot, as a rule, be treated by an operation. For these surgically inaccessible tumours, the internal administration of iodide of potassium has been found to be a specific remedy. Thomassen, who in 1885 was the first to recommend internal treatment with iodine, advises that each of the bovine sufferers should get daily, for 14 days, 1½ drams of iodide of potassium* dissolved in half a pint of water. The dose may be reduced to 1 to 1¼ drams when convalescence

* I am inclined to think that non-success in the treatment of actinomycosis by the internal administration of iodide of potassium has, in many cases, been due to a too sparing use of the drug. In one instance of a three-year-old thoroughbred colt which was suffering from actinomycosis of the tongue, and which had derived no benefit from a course of daily doses of half an ounce, I obtained a perfect cure by at first quadrupling the dose and giving during 38 days 52 ounces. When I first saw the colt, his tongue was so greatly swollen, and was so hard and painful from the actinomycotic changes in it, that he was unable to eat any kind of forage, and was consequently in the last stage of starvation. After 10 days, during which time I kept him alive by a plentiful supply of raw eggs, he was able to eat grass fairly well. The course of recovery was marked by the tongue becoming smaller, softer, and more mobile; by disappearance of the previous fœtid smell from the mouth; and by loosening of the actinomycotic nodules in the submucosa of the tongue, so that they could be readily shelled out with the finger-nail. On two occasions during this treatment, I discontinued the iodide of potassium for 3 days; but had to recommence it without further delay; for the symptoms of swelling and hardness of the tongue, running of saliva from the mouth, and adherence of the fibrous nodules to the tissues, began to rapidly increase, but abated with equal quickness under the renewed influence of the drug. The only untoward effect produced by these enormous doses was difficulty in staling, which appeared only after a month, and which soon passed off after the removal of its exciting cause. This colt took about 6 months to regain the use of his tongue sufficiently to enable him to eat oats efficiently. He regained perfect health, and was subsequently trained for racing.—Tr.

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sets in. It is stated that a visible improvement is noticeable after 8 days of this treatment, and that the affection becomes cured, on an average in a fortnight, if the disease be restricted to the tongue and neighbouring soft parts. Numerous trials have proved, with a few exceptions, the specific curative effect of iodide of potassium (Fürthmeyer, Bass, De Jong, Nocard, Ostertag, Deslex, Reeks, Perinni, Havas, Gooch, Soucail, Hohenleitner, Krug, Schwäbel, Walther, Salmon, Bang, and others). The internal treatment with iodide of potassium may be supplemented by external application of iodine in the form of the tincture, or of Lugol's solution, either of which can be applied by means of a brush or injected subcutaneously. Sometimes, also, a spontaneous shrivelling of the actinomycoma is observed to occur without any treatment. The mere incision on the actinomycotic tongue, without application of iodine, sometimes effects a complete cure (Bassi).

Actinomyces Musculorum Suis.—Besides the ordinary fungus of cattle and swine (actinomyces bovis), Duncker has found in the muscles of the pig a second variety of the fungi which is club-shaped and which radiates from the centre and occurs in the muscles only. Duncker and Hertwig have called this fungus "actinomyces musculorum suis" to distinguish it from "actinomyces bovis." These authors do not claim that this ray fungus is identical with actinomyces bovis, with which it has apparently no connection. It has been found in a large number of pigs, and forms, when magnified 40 to 50 times, grey-brown foci of round, distinctly defined corpuscles. When magnified 300 times, we find in these foci micrococci-like formations, between which club-shaped filaments lie. The neighbouring muscular tissue is diseased, and the flesh is soft, very watery, and is on that account loathsome and unfit for human consumption. Hertwig advances the conjecture that the germs of the fungus are ingested during the summer months or at the commencement of autumn. Johne distinguishes actinomyces musculorum suis from actinomyces bovis by the absence of the very characteristic blackberry-shaped tufts and by the fact that changes in the meat occur only in severe cases of the disease. According to Pfeiffer, we have to here deal with a myositis sarcosporidica, namely, infection by Miescher's tubes (sarco-sporidia). Olt is of opinion that it is a special form of muscular degeneration, produced by streptococci; while Davids holds that it is a simple waxy or hyaline degeneration, not caused by micro-organisms.

Actinomycosis of Man chiefly occurs in the form of a suppurating inflammation and abscesses. The bones of the face and the tongue are the chief points of attack. From them, other organs such as the lungs, liver, kidneys, peritoneum, intestines and brain become affected by metastasis. As in cattle, the infection is probably transmitted exclusively by portions of plants which are studded with fungi. According to German law for the regulations of the inspection of meat, only the altered portions of the flesh need be looked upon as unsuited for human consumption.

BOTRYOMYCOSIS IN HORSES.

Etiology. — By the term botryomycosis is indicated an infective disease, which is produced by the botryomyces fungus (botryococcus pyogenes, micrococcus ascoformans, micrococcus botryogenus), and is characterised by chronic inflammatory proliferations of connective tissue: so-called botryomycoma or mycofibroma. The fungus was discovered in 1884, and has been fully studied by Rivolta, Johne, Rabe, Bollinger, Jensen, Kitt, Hell, De Jong, Poncet and Dor, Parascandolo and others. It forms blackberry-like masses of round coccus threads. Although the identity of the botryomycosis-parasite with the ordinary pus coccus (staphylococcus pyogenes aureus), was formerly maintained (Hell, Kitt, De Jong), the specific nature of this fungus appears to have been established by the more recent researches of Parascandolo, Poncet, and Dor. The results of inoculation-experiments on horses testify against their identity; so do the various bacteriological, physiological, biological and sero-diagnostic relations of the two specimens of fungi. The botryomyces fungus can assume pyogenic properties; but the staphylococcus can never become botryogenic (*i.e.*, lead to the formation of myco-fibroma). The staphylococcus forms golden-yellow cultures at all temperatures; the botryomyces fungus only at 64.4° F. Bacteriologically, the two resemble each other in form, staining with anilin dyes, and grape-like arrangement; but their development on gelatine is different, and they display a remarkable divergence in power of immunisation. Only the pyogenic, not the botryogenic, properties are common to both.

Occurrence.—Botryomycosis occurs very frequently in the horse. It may, like actinomycosis, be regarded as a disease derived from wound-infection; as the botryomyces fungus usually finds entrance through small wounds of the skin; for instance, operation-wounds (castration). A generalisation of the infection occurs, however, less frequently than in actinomycosis. It is generally found in the skin and subcutaneous tissue, the udder, nasal mucous membrane and muscles, and more rarely in the bones. Besides occurring in the horse, it has occasionally been observed in cattle and in pigs, and recently also in human beings.

Symptoms.—Botryomycoma is most frequently met with

in the skin. It appears there partly as solitary, partly as multiple tumours distributed over the whole body; especially in the saddle and harness parts; at the cubital joint, tail, pastern, lips, eyelids, vicinity of parotid gland, hock, and scrotum. They vary in size frequently from a pea to that of a man's fist; often, however, they attain dimensions far larger than those of any other tumour. Thus there may be found at the cubital joint, in the neighbourhood of the shoulder, and at the pastern, tumours of many times the size of a man's head. Sometimes a great number of small nodules are found grouped round a large primary one. Their consistence is generally dense, but in some cases soft and even presenting a limited fluctuation; fistulous canals sometimes lead into the depths of the tumour. On section there is found a sand-like imbedding of botryomyces threads, as well as fluid spaces with soft granulation-tissue and small pus-cavities, in a solid, sinewy, sclerosed connective-tissue tumour. The large swellings grow from the skin into the subcutaneous tissue and the deeper organs.

Almost of as frequent occurrence is the botryomycosis of the spermatic cord. Most cases of the so-called fistula of the spermatic cord in horses are to be regarded as funiculitis botryomycotica, or botryomycoma of the spermatic cord. The infection of the castration-wound takes place from the straw. The stump of the spermatic cord begins to swell, and forms ultimately, in connection with the likewise chronically indurated common sheath, a dense, cylindrical tumour of size varying from that of a fist to that of a man's head; which reaches to the inguinal canal, and sometimes also displays fungous swellings between the margins of the wound (so-called mushrooms—"champignons"), and frequently encloses one or more fistulous channels which open beneath into one or more funnel-shaped fistulous openings. From these a discharge escapes containing the botryomyces fungus. In many cases, the botryomycoma also extends from the spermatic cord to the sheath of the penis, and over the neighbouring skin of the lower abdomen and thigh, so that enormous tumours—many times the size of a man's head—are developed. Often a bilateral fistula of the spermatic cord is formed. In amputated preparations, there are found on the lardaceous, dense, thready, grayish-white cut surface, yellowish-brown, flabby, muco-purulent masses of softening; in which the yellowish-white, grains-of-sand-like botryomyces particles may be seen with the naked eye.

Botryomycosis of the udder is common amongst mares (Möller, Sand, Vennerholm, Fröhner). The udder then displays swelling with dense, nodular induration; fistular openings, and surrounding abscess-formation. The typical botryomyces threads may be seen in the pus of the fistular channels. Unterhossel has described a botryomycotic new-growth on the udder of a mare which weighed 77 lbs.

A *myosotio botryomycotica* occurs especially in the sterno-cleido-mastoideus muscle, where it presents the form of a breast-boil or shoulder-boil; it has also been found in the abdominal, intercostal, and lumbar muscles. On the other hand, botryomycosis of the bones is very rare; in one case, described by Kitt, a rib was changed by a neighbouring botryomycoma into a spongy, osteoporotic mass. Storch has described a botryomycoma in the horse, which arose from the maxillary antrum. The projecting new growth arising from the mucous membrane had reached to the size of a child's head in two months, and produced an upheaval and asymmetry of the vicinity of the maxillary sinus and forehead. Cases of generalised botryomycosis have been described by Rieck, Fröhner, Tempel, Kitt, McFadyean, and others. It is very rare, in contrast to actinomycosis, and occurs especially in the lungs, peritoneum, heart, kidneys and other internal organs. Occasionally there has been observed in cattle an extension of botryomycosis of the lungs to the parietal pleura and costal wall; with osteitis granulosa, rarefaction and fistula formation in the ribs. Rabe has described a botryomycoma of the pelvis which weighed 33 lbs.; and had been complicated by the formation of a fistula, opening externally and communicating with the bladder.

Therapeutics.—Botryocomata, like actinomycomata, should be removed by operation—wherever possible, and without delay. There are cases, however, which on account of their enormous dimensions, or from the occurrence of generalisation, are no longer amenable to operation. Potassium iodide has been recommended as a specific against both actinomycosis and botryomycosis. Thomassen gives horses 2½ drs. to ½ oz. of potassium iodide daily, and treats the fistula of the spermatic cord with tincture of iodine locally. After a month's treatment the tumours have greatly diminished in size. Siegmund gave doses of 3 drs. to ½ oz. of potassium iodide three times a day; so that one horse consumed a total of 27 ozs., and another 40 ozs., from which he obtained good results

A correspondingly favourable report of potassium iodide has been given by Ostertag, Malkmus, and others. Our own (Fröhner) experiments and researches speak unfavourably of the curative power of potassium iodide. In the case of a horse affected with botryomycosis of the spermatic cord, skin, abdominal muscles, and lymphatic glands, neither the internal administration of ¾ lb. of potassium iodide, nor the subsequent intratracheal injection of 7 ozs. of sodium iodate, produced any improvement—much less a cure. The morbid process had extended very largely, in spite of the iodide treatment. Another horse affected with botryomycosis of the spermatic cord received, during 5 months, a total of 25 ozs. of potassium iodide, without any apparent beneficial result. On the other hand, the symptoms of chronic iodism developed very early in the animal. He was then operated on, and recovered rapidly. Similar misadventures with potassium iodide in the treatment of botryomycosis of the udder have been recorded by Vennerholm, König, and Winter. Töpper has also pronounced generally against the treatment with potassium iodide.

Botryomycosis in Man.—In recent years many cases have also been observed in human beings. Poncet and Dor have four times found in man botryomycotic tumours on the fingers, thorax, and elbows; and successfully inoculated a donkey with the disease (French Surgical Congress, 1897). Ten Siethof saw conjunctivitis palpebrarum of botryomycotic nature, and accompanied by nodular formations, develop in a man who had had charge of a horse suffering from fistula of the spermatic cord. Other cases have been described by Legrain, Sabrezès, Laubié, Delor, Galli-Valerio and Lenormand.

STOMATITIS PUSTULOSA CONTAGIOSA OF HORSES.

Nature.—Stomatitis pustulosa contagiosa of the horse is an infective acute exanthema which chiefly attacks the mucous membrane of the mouth in the form of pustules and is distinguished by a benign course.

History:—From a careful study of veterinary literature, we find that stomatitis pustulosa contagiosa is by no means of recent date. Dard described, as early as 1840, a vesicular inflammation of the nasal mucous membrane which he had noticed in 1832 among 15 horses of the same battery, and

had at first considered it to be acute glanders. The description of the disease, which lasts on an average 20 days and is non-malignant, leaves nothing to be desired. Bouley, Patty, Reynal, and Jakob reported exactly similar cases between 1840 and 1850. In 1856 Hering described in a voluminous treatise an epizoötic outbreak of this exanthema, discussed its differential diagnosis with respect to acute glanders, and gave an account of inoculation experiments which he had made. About the same time König described the disease as aphthæ ulcerosæ of an apparently infectious character. In 1863, Vimercati reported on an equine mouth disease, by which 20 out of 90 cavalry remounts were attacked by a pustular dermatitis, stomatitis, rhinitis, and conjunctivitis. The publications of Palat and Silvestri on horse-pox undoubtedly referred to the same malady. Silvestri identified the complaint which he called "pox" as impetigo labialis of the German veterinary surgeons. The observations of Sondermann, Immelmann, Eggeling, and Haarstick refer indisputably to it. This disease gained renewed interest in 1878 by a publication of Eggeling and Ellenberger, who proposed to term it stomatitis pustulosa contagiosa.

Etiology.—Stomatitis pustulosa contagiosa, as the name implies, is an extremely infectious disease. The contagium is fixed and is contained chiefly in the saliva and mucus, and consequently the mucous membranes are far more susceptible to the action of the virus than the cutis. As a rule, it penetrates the tissues only at the site of a previous injury; though it may also be transmitted to horses by mere contact or by an intermediate bearer. In the order of comparative susceptibility, the horse comes first, then cattle, men, sheep, and pigs. Birds also contract the disease. Men occasionally become infected, especially attendants, veterinary surgeons, and children, on the hands, arms, conjunctiva, etc. The virulence of the contagium seems to weaken by inoculation from animal to animal. As a specific fungus has not yet been found to account for this disease, more exact researches are desirable. A previous attack appears to confer immunity on horses.

Anatomical Conditions.—The initial change consists in an inflammatory swelling of, and infiltration of small cells into the papillary layer of the skin, by which a microscopic formation of nodules takes place. In the further progress

of the disease, the nodules suppurate and liquefy in their centre; the papillæ are destroyed, and the entire thickness of the skin or mucous membrane, and even the subcutis or submucosa may become involved. If, later on, a pustule which has sprung up in this way, opens, we shall find the tissue that surrounds the destroyed substance to be inflammatorily infiltrated. The healing of the ulcer takes place by the formation of granulations from below and by a new growth of epithelium from the edges. The cicatrix varies in size. About 12 days are required for complete recovery.

The foregoing changes are found chiefly on the mucous membrane of the oral cavity, namely, on the dental surface of the lips, and on the frænum, tip, and sides of the tongue. They also attack the cutis and mucous membrane of the nasal cavities. The ulcers may be isolated, or may become confluent so as to cover a large area. If the process proceeds to the conjunctiva, a purulent conjunctival catarrh is usually the only unfavourable consequence. In rare cases, the formation of nodules on the mucous membranes is preceded by petechiæ. Vesicles, instead of nodules, may appear as forerunners of the pustules.

Symptoms.—With slight premonitory fever (temperature up to 103° F., and rate of pulse up to 60 a minute), the mucous membrane of the mouth becomes intensely red and hot, and isolated nodules are felt on passing the finger over its surface. Gradually, masses of mucus accumulate in the mouth, profuse salivation takes place, and the animal resists exploration of the mouth. As a rule, the general health is but little disturbed. The appetite is unaffected at first; but later on, prehension of food becomes painful, and is effected with considerable salivation and even regurgitation. The temperature is seldom higher than from 104° to 106° F. After 2 or 3 days, the nodules increase in number and size, and their summits become pale and manifest the first signs of ulceration. In 4 or 5 days, the mucous membrane of the mouth gets covered with ulcers in various stages of development. On the mucous membrane of the lips, tongue, and gums, we find conical prominences, which are 0.07 or 0.11 inches high, and which are either isolated or in groups of from 2 to 7 or more in number. The apices of these prominences become always changed into circular ulcers. We can recognise in the ulcers a slightly depressed centre, which is excessively hyperæmic, shining, and

finely granulated. The ulcers are surrounded with a dirty-white or yellowish-white ring which looks like scalded epithelium, and which can usually be wiped off only with difficulty. By its removal, we expose the base of the ulcer, which readily bleeds, and manifests the condition that was first observed in the centre of the undisturbed ulcer. We also find larger isolated ulcers and collections of confluent ulcers which frequently extend into the submucosa. Similar changes sometimes take place in the skin and especially on the upper lip, cheeks, margins of the *alæ nasi*, nasal mucous membrane, and even on more distant regions of the body, as for instance, on the skin of the fore-legs, and anus (infection transmitted by sponging), in which case it may happen that even the oral mucous membrane remains intact. Besides these local affections, there is usually great congestion of the mucous membrane of the nostrils, and of the conjunctiva, which may exhibit an intense purulent catarrh. There is at the same time, great swelling of the neighbouring lymph glands, particularly of the submaxillary glands and even of the glands of the shoulders.

The duration of the disease varies according to the intensity of the attack. The ulcers often begin to heal after a week has elapsed. The average duration of the entire course of the complaint is about a fortnight, with a maximum of about 3 weeks.

Differential Diagnosis.—It is not always easy to distinguish stomatitis pustulosa contagiosa from other somewhat similar processes; although in no case is the task impossible, if all the factors are taken into consideration. We should especially bear in mind the following diseases:—

1. *Acute Glanders and Farcy.*—Glanders (using the term as inclusive of farcy) has been frequently mistaken for the disease we are considering. Hering, 30 years ago, pointed out some differential diagnostic points between the two complaints. A mistake may be easily made, when, besides ulcers on the nasal mucous membrane and skin, and a nasal discharge, we find hard, painless swellings of the neighbouring glands; and when, by chance, no pustular changes of the oral mucous membrane are present, although the action of a contagium is evident. It is probable that formerly many horses affected with stomatitis pustulosa contagiosa fell victims to this diagnostic error. The following points distinguish stomatitis pustulosa contagiosa from glanders: the entire course of the

disease is benign; the symptoms of stomatitis are not those of glanders; the cutaneous ulcers of pustular stomatitis are not confined to the course of the lymphatics, as in farcy, the characteristic, rosary-like ulcers of which, with their indurated and eroded edges, are also absent; and the ulcers are circular, isolated, and frequently covered with a firm crust, under which they show healthy granulations and signs of a growth of epidermis commencing at their edge. These ulcers heal in about a week. The ulcers on the nasal mucous membrane are found only at the entrance of the nostrils and possess the same distinguishing differences, as regards glanders, as those of the skin, and inoculation produces stomatitis pustulosa, but not glanders. Also, the disease can be easily transmitted to cattle, especially to the mucous membrane of the vagina.

2. *Horse-pox.*—Next to glanders, horse-pox has probably been more frequently confused with stomatitis pustulosa than any other disease. Pustular stomatitis more closely resembles true equine variola, both clinically and anatomically, than any other disease, and especially any other exanthema. We must bear in mind, as an important diagnostic distinction, that equine variola is exceedingly rare, and that it occurs as a rule only on the pasterns.

3. *Follicular Ulceration.*—Formerly, under this term, stomatitis pustulosa was frequently mentioned. We may point out that the ulcers of stomatitis pustulosa are not connected with the anatomical position of the follicles, and may occur on places in which there are neither glands nor follicles.

4. *Stomatitis catarrhalis aphthosa, traumatica et ulcerosa* attack neither the skin nor the nasal mucous membrane; they manifest neither swelling of the lymph glands, nor the typical uniformity of stomatitis pustulosa; and they are not contagious.

5. *Vesicular eruption on the genitals and dourine* need scarcely be considered; for stomatitis pustulosa up to the present has produced its characteristic changes on the mucous membrane of the genital organs, only after artificial inoculation.

6. *Herpes Labialis* shows neither pustules nor ulcers.

7. *Canadian Horse-pox* is localised as a rule on the back (region of the saddle).

Prognosis and Therapeutics.—Prognosis is wholly favourable; because no recorded case has up to now ended fatally. For instance, about 1,000 horses became affected in the Prussian

army from 1887 to 1889 without any loss. As it, like all acute exanthemata, runs a typical course, no special treatment is necessary; although we may disinfect the ulcers with solutions of creolin, carbolic acid, alum, sulphate of iron, chlorate of potash, etc. As the disease is intensely contagious; it goes almost without saying that prophylactic measures should on no account be neglected.

DIPHTHERITIC DISEASES OF DOMESTIC ANIMALS.

General remarks—General remarks on diphtheritis of animals and diphtheria of man—Diphtheria in man—Croupous diphtheritic inflammation of the mucous membranes in fowl—Avian croupous diphtheritic inflammation of the mucous membrane probably caused by schizomycetes—Avian croupous diphtheritic inflammation of the mucous membrane caused by gregarines—Diphtheria of calves—Diphtheria of pigs.

General Remarks.—The name of diphtheritis is given to a particular form of inflammation of the mucous membrane which is characterised anatomically by a fibrinous exudation in the interior of the tissue with consequent necrosis, that is to say, a combination of exudation and mortification. By this definition we distinguish diphtheritic inflammation from croupy inflammation, which consists only of a superficial fibrinous inflammation of the mucous membranes with formation of croupy membranes that can be peeled off, without the deeper lying portions of the mucous membrane being implicated.

Diphtheritis of the mucous membranes is by no means a clinical unit; for it can be produced in animals by greatly-varying causes. Thus it may spring from purely mechanical means through pressure, as for instance, fæcal accumulation. Very many chemical substances, especially caustics, produce in the mucous membranes a form of a diphtheritic necrosis. A large series of infectious agents may also set up diphtheritic inflammation. We accordingly find diphtheritic processes during the course of rinderpest, malignant catarrhal fever in cattle, swine fever, acute glanders, petechial fever, "snuffling" disease of pigs (*Schnüffelkrankheit**), septicæmia

* The word *Schnüffelkrankheit* appears to be used as a general term for difficulty of breathing due to stenosis of the nasal passages, whether caused by infectious rhinitis or rachitis.—Tr.

puerperalis, gregarinosis of birds, and certain infectious diseases, such as diphtheritis of the mucous membrane of the nose and of the intestine, the causes of which, up to the present, have not been elucidated. Diphtheritic local affections may be produced experimentally in animals by the inoculation of different bacteria, especially the necrosis bacillus.

The same great variety in the clinical conception of this disease also exists with regard to human diphtheria. Therefore, in human medicine, a distinction is made between primary and secondary diphtheritis. The term primary diphtheritis or true diphtheria is applied to the specific, independent infectious disease, which is characterised by diphtheritic inflammation of the mucous membrane of the pharynx and which is produced by Löffler's diphtheria bacillus. Diphtheritic pharyngitis may occur secondarily to other infective diseases, such as scarlet fever, measles, smallpox, erysipelas, and whooping-cough, and is then called secondary diphtheritis or pseudo-diphtheria.

General Remarks on Diphtheritis of Animals and Diphtheria of Man.—The diphtheritic diseases of domestic animals are in no way related to human diphtheria. No indisputable case of the transmission of diphtheria to man from an animal has been proved. The cases recorded in medical literature, of the alleged transmission of infection, especially those of chicken-diphtheritis supposed to have been conveyed to man, are, on closer examination, reduced to mere assumptions, the forming of which has been due to entire ignorance of veterinary pathology. Even the statement of Gerhardt —which is the only one of the kind worthy of being quoted —that on a poultry farm, two-thirds of all the workmen attending on the fowl which were suffering from diphtheritis, became affected with pharyngeal diphtheritis, cannot be substantiated; for Gerhardt did not observe the case personally. Considering the wide dissemination of chicken-diphtheritis, especially in poultry farms, and presuming that this disease is identical with human diphtheria, a very great number of people would necessarily become daily infected, and reports on such cases would not be, as they are, extremely rare. We have examined thousands of domestic fowl and pigeons suffering from diphtheritis without having either seen or heard of a single case of such infection. We have also the results of the bacteriological researches carried out in great numbers by Guérin, Barella, Gratia, Liénaux, and others; and the absolutely

negative results of the treatment of many sick fowls with human diphtheria serum—all testify against the identity of the two diseases. No indisputable proof has yet been given that any disease similar to human diphtheria has been transmitted, either experimentally or accidentally, to any of the domestic animals, especially horses, cattle and dogs.

Diphtheria in Man.—Contrary to the pathological nature of the diphtheritic ailments of domestic animals, which will be discussed later on under the headings of chicken-diphtheritis and calf-diphtheritis, human diphtheria is a specific acute infective disease which manifests itself locally in the form of a croupy diphtheritic inflammation of the mucous membranes, especially those of the pharynx and larynx. Löffler regards specific bacilli as the cause of human diphtheria. His diphtheria bacilli are stout rods about the length of a tubercle bacillus, double its thickness, and with rounded ends. They are found only in diphtheritic membranes. Their toxins become absorbed into the blood and produce, by means of it, serious general phenomena. Cultures of Loffler's bacilli are said to produce in rabbits and pigeons, croupy membranes in the trachea, and sometimes grave constitutional disturbances and paralysis. Inoculated guinea-pigs die in a few days under symptoms of exudative pleuritis and œdema. The disease occurs chiefly among children up to their tenth year; it is very contagious; and begins probably as a local pharyngeal affection. After an incubatory period of from 2 to 5 days, symptoms of general disturbance (fever, malaise, and headache) first of all appear, and then difficulty in swallowing. The mucous membrane of the soft palate becomes greatly congested and the tonsils swollen. Dirty-white, firmly-attached covering membranes soon form on the mucous membrane of the pharynx (tonsils, uvula, and the palatine arch) with considerable swelling of the neighbouring lymph glands. The diphtheritic process then spreads to the mucous membrane of the larynx, where it causes stenosis of the larynx (dyspnœa and suffocation), and even to the mucous membrane of the bronchi and bronchial tubes. The symptoms of a septic general infection are also present. The chief complications are as follows: extension of the diphtheritic inflammation to the nasal cavity, oral cavity, tympanic cavity, conjunctiva, and intestinal mucous membrane; swelling of the articulations; myocarditis; nephritis; paralysis of the soft palate, vocal chords, muscles of the eye, muscles of deglutition, and, less frequently, muscles of the extremities; and ataxy. The mortality is very high. Fränkel, followed by Behring, was the first to begin the experiments which have been recently made for obtaining immunity against, and for finding a remedy for, diphtheria. Behring succeeded in curing, by the injection of the terchloride of iodine, guinea-pigs which had previously been given a lethal dose of diphtheria cultivations. The blood serum of these infected animals proved itself to be an absolutely certain agent for conferring immunity. Recently, Behring has begun to use horses for the manufacture of his diphtheria serum.

I.—Croupous Diphtheritic Inflammation of the Mucous Membranes in Fowl.

General Remarks.—The croupous diphtheritic inflammation of the mucous membranes in domestic fowl is not an etiological unit. On the contrary, we must distinguish at least two etiologically different varieties, which, although they possess very similar symptoms, are nevertheless different in their nature. They may be described as follows: (1) a croupous diphtheritic inflammation of the mucous membranes, caused most probably by bacteria; (2) a croupous diphtheritic inflammation of the mucous membranes caused by gregarines.*

Independently of microscopic examination, the croupous diphtheritic stomatitis which is produced by gregarines may be distinguished from that due to bacteria, by the fact that it can be transmitted by inoculation only with much difficulty. In spite of the great contagious influence of cohabitation, the disease has been directly transmitted by inoculation only in a very few cases. The inflammation of the mucous membranes caused by gregarines can, however, be transmitted by inoculation with the utmost facility.

A.—Diphtheria of Fowls.

Croupous Diphtheritic Inflammation of the Mucous Membranes caused by Schizomycetes.

Etiology.—Speaking generally, this form is, next to typhoid, the most common and most dangerous epizoötic among fowl. It has become generally known only during the last 30 years. Leisering, in 1860, appears to be the first veterinary author who described it. Zürn states that Russ in 1861 was the first to describe chicken diphtheritis. The existence of numerous publications by Italian and French veterinary surgeons referring to this disease, and other facts which we shall presently discuss, indicate that its starting point was in Italy and France, from which countries it was imported into Germany.

This affection was formerly considered by many to be of a tuberculous nature; but later on, it was regarded as true croup true diphtheritis, or a croupy diphtheritic inflammation. As the fact had been proved that bacteria were frequently found in the

* Single-celled parasites which belong to the sporozoa.—Tr.

inflammatory products, the view was generally adopted that these fission fungi possessed a pathogenic importance: a supposition which appears to be correct. Recently, various more or less contradictory theories have been advanced about other forms of fungi. Rivolta refers the cause of chicken-diphtheritis to two distinct species of fungi, which he calls "epitheliomyces croupogenus" and which develop only in the skin and mucous membranes, but not in the tissues or blood; thus entirely differing from the cocco-bacterial affection of the blood in diphtheria of man. In cases of croupy diphtheritic inflammation of the mucous membranes of pigeons, Löffler found in the different exudates and in the liver, numerous bacilli which were somewhat longer and thinner than those of the septicæmia of rabbits and which had rounded edges, besides several other kinds of fission fungi. Subcutaneous injections of pure culture of these rods produced necrotic inflammation, and in mice a characteristic infective disease. By the retro-inoculation of a pure cultivation which was obtained from the liver of a mouse, into two pigeons, diphtheritis of the oral cavity was produced in them. Löffler believes, however, that these bacilli should be demonstrated in a series of cases, before they can be looked upon as the true cause of pigeon-diphtheritis. According to Guérin and Nocard, the diphtheria of fowls is produced by a cocco-bacillus of the Pasteurella group.

Bacteriology.—Eberlein constantly found in diseased partridges, chain-like bacilli from 2 to 5 μ long and from 1 to 2 μ broad, which were rounded off at their corners and which contained spores. Ritter proved the presence of the Löffler bacillus in a case of fowl-diphtheria, and was able easily to infect healthy birds with cultures of it. He thinks that the necrosis bacillus plays a part in this disease. Moore found a bacillus, very like that of Löffler, but not communicable by inoculation. Ferré saw in the diphtheria of fowls, the same species of schizomycetes as in men: micrococci, staphylococci, streptococci, pneumonia bacilli, colon bacilli, and the Löffler bacillus. On the results of their own experiments Gratia and Liénaux pronounce against the identity of fowl-diphtheria and human diphtheria (cultures different; absence of effect of the diphtheria serum of Behring and Roux in affected fowls and pigeons). Guérin has arrived at the same result (bacteriological investigation of 78 cases of the disease; negative serum treatment of 200 affected fowls). Barelle also has shown, by numerous experiments, that the diphtheria of men has nothing in common with that of fowls. Doroschanko has carried out researches on the immunisation of fowls and pigeons by means of sterilised bacilli cultures, and found that the immunity artificially obtained lasted from 4 to 5 months.

Occurrence.—The inflammation of the mucous membranes

caused by bacteria occurs, except in rare cases, only as an epizoötic and attacks chiefly fowl and pigeons of the finer breeds, particularly those imported from Italy and France. German country fowl rarely suffer. Young birds are more liable to become infected than old ones. Parrots and water-fowl are attacked as well as gallinaceous birds (domestic fowl, turkeys, guinea-fowl, partridges, peacocks, pheasants) and pigeons. Fowl-diphtheria sometimes assumes the character of a very severe epidemic. For instance, in Baden, there were two outbreaks in 1903, in one of which (at Brötzingen) 1,300 fowls died, and in the other (at Nesselhausen), of 2,600 which had been brought from Italy, 1,400 and nearly 1,000 breeding hens also perished. According to Lang, fowl-diphtheria reached so destructive a point in New Caledonia, that the rearing of fowls became excessively difficult.

The chief cause of the large increase of the disease is the modern taste for fancy breeds, which has greatly stimulated the importation of foreign birds. Poultry shows, which have become more and more fashionable, certainly aid in the distribution of this malady; because sufficiently severe prophylactic regulations are not always applied to their management.

Symptoms.—Hardly any disease is so polymorphous as croupy diphtheritic inflammation of the mucous membranes caused by bacteria. Sometimes the mucous membrane of the mouth and pharynx is attacked; at other times, that of the nostrils and lining membranes of the accessory cavities. The affection may be limited to the mucous membranes of the eyelids and eyeball, or it may spread from the mouth into the larynx, bronchi, and alveoli of the lungs. In other cases, the alimentary canal may be the chief seat of the malady. The skin may also become involved. These respective local manifestations may appear singly or combined. The period of incubation lasts for a few days.

1. *General symptoms.*—The general health, especially in mature, strong animals, is but little disturbed at the commencement of the disease. Differing from the course of human diphtheria, the local changes may be fairly well advanced before the illness of the patient attracts attention. We desire to lay particular stress on the fact, which has been denied by other writers, that the internal temperature, even when the symptoms are severe, never rises to an inordinate height, either at the beginning of the disease or during its progress. Anomalies in

the distribution of the blood, defective nutrition and emaciation with their consequences, are to be observed during the further progress of the complaint. Thus we find that the collapsed erectile tissue on the head (comb and wattles) is dry and of varying temperature; the superficial heat of the body is unequally distributed; the visible mucous membranes are pale; and the feathers ruffled. The bird is off its feed; it gradually ceases to lay if it is a hen; it becomes depressed in spirits and feeble; droops its wings; makes only a feeble resistance when taken hold of; and likes to isolate itself. Before the fatal ending of the disease, the internal temperature sometimes falls, and the patient shows slight symptoms of brain irritation, quickly followed by cerebral depression.

2. *Croupous diphtheritic inflammation of the mucous membranes of the mouth and pharynx.* The first symptoms, which usually escape notice, are hyperæmia, and a slight swelling of the mucous membrane, which very quickly gives place to a deposit resembling hoar frost in appearance. This covering, which is thin at first, perceptibly increases in thickness, generally within 24 hours, and gradually changes into a white, fairly even, shining, caseous substance of a rather tough consistence, and very firmly adherent to its base. The thickness of this deposit, which soon assumes the aspect of a pseudo-membrane, may vary from 0.04 to 0.06 inches. Its colour gradually changes to dirty yellow, and later on to brown. Its surface becomes rough and fissured, and its consistence, dry and brittle. The bird is usually obliged to breathe with an open beak, the head and neck being stretched out, and inspiration and expiration are very laborious. Prehension of food and deglutition are more or less seriously impaired. The favourite seats of the attack, which is not distributed over the entire surface of the mucous membrane, are the soft and hard palate with the cleft of the palate; inferior surface of the tongue with the frænum and tip of the tongue; inner surface of the cheeks; angles of the mouth; and especially the neighbourhood of the superior portion of the larynx. From the angles of the mouth, the affection usually spreads to the skin. When the deposit has been removed—which, as we have already said, can be done only with difficulty—there always remains a more or less deep, uneven, greatly reddened or bleeding ulcer with eroded edges. We may notice at the base of this ulcer, various fine, villous proliferations, in which case isolated vascular loops project into the plates of the exudation, the removal of which gives

rise to hæmorrhage. In the further progress of the disease, either the deposit becomes detached and a perfectly intact mucous membrane is then exposed to view, or ulcers resembling hard chancres, form, with probable loss of tissue and even necrosis, as for instance, that of the tip of the tongue.

3. *Croupous diphtheritic inflammation of the mucous membranes of the nasal cavity and neighbouring cavities.* At the beginning of the attack, we find in the neighbourhood of the nostrils, a serous fluid, which later on becomes dirty-yellow, greasy, and dries up, and then partly plugs up the nostrils and nasal ducts. If we press the nasal cartilages, we shall obtain the discharge of a fluid which is at first serous, and later on, more or less slimy, and is finally of a milky-purulent character. As a consequence of this discharge, the respiration becomes laborious and snuffling. The bird sneezes, by doing which, mucus is discharged, and at the same time, the patient frequently shakes its head. We find in the cleft of the palate inflammatory products, which we have already described. The process then very frequently spreads to the lining membrane of the cella infraocularis, which is more often unilaterally than bilaterally affected. Under the median angle of the eye and behind the root of the beak, there is a protuberance of the soft parts, which is hyperæmic and collaterally œdematous, painful to the touch, and generally hot. Pressure on it produces an abundant discharge from the nostril of that side. This swelling goes on increasing, spreads chiefly under the eyeball and towards the rear, and may finally attain the size of half a walnut. At the same time, the corresponding half of the hard palate increases to at least double its width, and bulges out into the oral cavity. In its further progress, the tumour over the cella infraocularis becomes tense and firm. If opened early it will discharge a thick, creamy fluid, or a soft, cheesy substance. Later on, the dirty-white or yellowish contents become dry and crumbling, or present the appearance of tough, thin membranes or flakes lying closely together. By enormous accumulations of the dried-up exudate, the before-mentioned sinus frequently becomes dilated to such an extent that it may attain a diameter of $\frac{3}{8}$ to $1\frac{1}{8}$ or even $1\frac{3}{8}$ inches. The continually-increasing pressure causes displacement of the neighbouring soft parts, especially of the eyeball, other half of the palate, etc.; and osseous atrophy with absorption, in consequence of which the head becomes greatly disfigured and misshapen. The bird is often hardly able to open its beak, or to feed.

4. *Croupous diphtheritic inflammation of the mucous membranes of the larynx and trachea.* Considerable exudative accumulations take place in the neighbourhood of the opening of the larynx on the mucous membrane which lines the larynx, and on the upper part of the trachea, which can easily be examined in birds, especially in large domestic fowl; the resulting dyspnœa being proportionate to the degree of stenosis. The breathing is deep and laborious; the air being, as it were, pumped up; and the beak is held wide open. The bird gives utterance to peculiar whistling, singing, or wheezing noises, combined with moist râles, coughing, and panting, and frequently dies of suffocation. The coughed-up matters partly accumulate in the pharynx and partly adhere to and soil the throat and breast. Their decomposition gives rise to the disagreeable, sickly smell which we may perceive, even when at a considerable distance from the affected birds.

5. *Croupous diphtheritic inflammation of the eyes.* Catarrh of the eyelids is the first symptom of this complaint. The initial hyperæmia of the conjunctiva soon changes into a muco-purulent catarrh, the discharge of which collects at the median angle of the eye and flows away. The eyelids and their neighbouring parts become œdematous and hot; and the eyelids have a great tendency to stick together. By forcibly pulling them open, vent is given to masses of inflammatory products, which are at first of a serous and muco-purulent nature, but soon become caseous and then assume (corresponding to the form of the sac made by the eyelid and eyeball) a semi-lunar or lenticular shape. If the eye is left to itself, either the eyeball becomes atrophied by the pressure of the accumulated discharge, or, as very often happens, the diphtheritic process spreads to the internal parts of the eye and causes in it severe destructive changes (panophthalmia). At first, the disease spreads from the conjunctiva scleræ, to the cornea, which becomes affected by a superficial, smoky opacity, and which subsequently becomes swollen and opaque from parenchymatous inflammation (keratitis parenchymatosa), in which case a firm cone of exudation of varying size often quickly forms on the thickened, and now perfectly opaque cornea, and presses the eyelids asunder. Finally, suppurative perforation of the cornea and purulent panophthalmia with subsequent destruction of the eyeball, occasionally take place.

6. *Intestinal affection due to croupous diphtheritic inflammation of the mucous membranes.* This affection of the intestinal

canal consists in a more or less extensive catarrhal inflammation and croupy diphtheritic infiltration, which, as a rule, occurs only when one of the previously described local maladies has been present for a considerable time, and usually ends the course of the disease. Zürn states that it occurs early, as an independent complaint, in water-fowl and turkeys. In addition to an increase in all the general symptoms, diarrhœa sets in ; the fæces being fœtid, very thin, pappy or mucilaginous, and sometimes mixed with blood or bloody pus. Zürn states that this diarrhœa causes excessive debility, dulness, and stupor, and, when it appears in a severe form, is a forerunner of death.

7. *Affection of the skin in croupous diphtheritic inflammation of the mucous membranes.* The transmission of the croupy diphtheritic affection to the skin appears on the eyelids and corners of the mouth only to a small extent in the form of cutaneous ulcerations. Similar changes may also be found in the neighbourhood of the anus and on the lining membrane of the auditory meatus.

Post-mortem Conditions.—The *post-mortem* examination of the usually emaciated and anæmic cadaver sometimes shows, besides the already described changes which could be demonstrated during life, accumulations of whitish-yellow secretion in the bronchi, which appear to be filled with firm, crumbling masses, whilst the parenchyma of the lungs manifest signs of atelectasis or œdema. More frequently we find simple tracheal and bronchial catarrh with more or less extensive catarrhal inflammation of the lungs. Cellular fibrinous pericarditis, subepicardial ecchymoses and cloudy swelling of different organs, such as the liver, are by no means rare. The catarrhal inflammation of the intestinal canal is often confined to the upper and anterior parts. Zürn sometimes found the cæcum and rectum completely filled with yellow, stratified masses which adhered firmly to the intestinal mucous membrane ; and that the intestinal mucous membrane was frequently ulcerated. All authorities are agreed on the results of the microscopical examination of the excreted matters.

Microscopic Conditions.—The soft caseous discharge from the conjunctiva consists, for the greater part, only of round cells which cannot be distinguished in any way from degenerated leucocytes. They vary in length from 0.006 to 0.009 mm. and are distinguished by their great tendency to become rapidly broken up. Usually, a large number of bacteria, chiefly micrococci, are also present. When the inflammatory products

are treated with a watery solution of methylene violet, the uniformly small micrococci may be seen in enormous numbers. The rod forms seem to be less frequently present. The same result is obtained by the examination of the more recent aggregations in the mouth and pharynx, except that pavement epithelium in the exudate is more abundant in them and in the cavities of the head, bronchi, etc. The before-mentioned round cells quickly change, even in quite freshly excreted masses, into a fine granular detritus, so that we find an altered appearance corresponding to that found in the older masses.

Course.—The course of this croupy diphtheritic inflammation of the mucous membranes is always slow, and generally chronic. It usually lasts for weeks and even months, until the affected birds succumb to the disease. Only young birds, especially pigeons, form an exception to this rule; for they die of the disease in a short time. These facts agree with the few inoculation experiments which, up to the present, have been successful. Trinchera observed after inoculation with nasal mucus that the period of incubation was from 7 to 20 days, and that the symptoms slowly increased in intensity for from 8 to 15 days. He found that strong animals recovered their health in from 60 to 70 days. Krajewski states that the period of incubation in domestic fowl is from 4 to 10 days. According to the experiments of Babes and Puscarin, it lasts only 2 or 3 days in pigeons.

Prognosis is generally unfavourable. The comparatively hopeful cases are those of pigeons; of acclimatised, strong, well-nourished fowl; and of birds in which the process is limited to the pharynx. As a rule the disease is difficult to successfully combat, and supposed recoveries are often only apparent. Thus it happens that birds which are regarded as cured may, on being returned to their usual haunts, prove a cause of infection to their healthy companions, and the disease consequently acquires an insidious character. The average mortality is from 50 to 70 per cent.

Therapeutics.—*Prophylaxis* plays an important part in the croupy diphtheritic inflammation of the mucous membranes. It consists in exact examination; maintenance of quarantine for several days' duration with every newly-bought bird; abstaining from exhibiting birds at those poultry shows which are not under veterinary superintendence; carefully observing each bird; paying attention to suspicious symptoms, especially nasal discharge, tears, etc.; segregation of the affected from the

healthy; scrupulous cleanliness in coops and cages; periodical disinfection with carbolic acid, corrosive sublimate, creolin, lysol, etc.; and destruction of the bodies of birds which have died from the disease, and of the utensils that have come in contact with them. A veterinary police campaign against fowl-diphtheria must at present be postponed, as the unity of the disease is doubtful (the variola of fowls must always be separated from it). Besides, statistical data regarding the occurrence of epidemics of the disorder are wanting.

If in spite of the foregoing precautions, the disease has once broken out, its course may be favourably influenced by early treatment. Careful nursing is more efficacious than medicinal agents, among which we may mention the following disinfectants: carbolic acid, creosote, tar, creolin, lysol, chlorate of potash, permanganate of potash, corrosive sublimate, chloride of lime, tincture of iodine, salicylic acid, boric acid, sulphate of iron, liquor ferri perchloridi, sulphate of zinc, tannin, oil of turpentine, caustic potash, nitrate of silver, lactic acid, etc. These have been used either singly or in various combinations.

We have found the application by a brush, of a solution of corrosive sublimate (1 or 2 in 1,000), or one of creolin (1 or 2 in 100), to be very effective when the disease is localised. Other practitioners have also frequently obtained good results with creolin. We can recommend the removal of the pseudo-membranous inflammatory products, especially in the case of pigeons, only when it can be done easily, and without causing hæmorrhage. Our own experience prompts us to deprecate forcible removal and cauterisation of the base of the ulcer. The tumours over the cavities of the head should be incised, their contents removed, and the frequently severe hæmorrhage prevented by inserting a cotton-wool plug saturated with liquor ferri perchlor. We have found fumigations with tar especially effective in relieving the affection of the air passages. Zürn recommends internally and externally a mixture of about 5 oz. of a decoction of walnut leaves (½ oz. to 2 pints of water), with ⅝ oz. of glycerine, 1½ drams of chlorate of potash, and 7½ grains of salicylic acid in ½ oz. of rectified spirit. Of this we may give once or twice daily, from a teaspoon to a tablespoonful to large birds; and from a quarter to half a teaspoonful to pigeons. We should also paint the parts with this solution twice or thrice daily. For the intestinal complaint we recommend tannin or sulphate of iron (in a 1 to 2 per cent. solution or in pills made up with butter and white bread; for pigeons 3 to 7½ grains;

fowls $7\frac{1}{2}$ to 15 grains; and geese $7\frac{1}{2}$ to 30 grains *pro dosi*). The employment of diphtheria serum in the treatment of affected fowls has not proved satisfactory. Richard's prescription has the following composition: ℞. calc. chloridi. 2 drs.; acidi salicylici, $\frac{1}{2}$ drm.; glycerini, $4\frac{1}{2}$ drs.; aquæ succo. viridi tinctæ, 4 oz. 5 drs. M.D.S. To be used for brushing the cavity of the mouth.

B.—Fowl-pock.

Croupous-diphtheritic Inflammation of the Mucous Membranes and Skin caused by Disease-agents capable of Filtration.

Etiology.—Fowl-pox (epithelioma contagiosum) is an infectious and specific disease of the skin, and mucous membrane, which occurs in fowls, geese and pigeons, and the cause of which was formerly attributed to gregarines. This view, which was first promulgated by Rivolta in 1872, was advocated by us in the first five editions of our text-book, in accordance with the then existing knowledge on the question. The recent researches of Marx and Sticker (1902), prove that the particles formerly referred to as *gregarines*, which are enclosed in the epithelium (so-called epithelial corpuscles, molluscum corpuscles, Guarnier's corpuscles) are no other than products of epithelial metamorphosis, which have been observed in other diseases (variola and molluscum contagiosum in man). Thus the other parasites hitherto regarded as the causative agents of fowl-pox can no longer be considered. The infective material is still unknown. Bacteriological researches have given a negative result. The researches of Marx and Stickler have, however, shown that the causative agents in fowl-pox belong to the group of so-called filtrable agents of infection, to which also the agents of foot-and-mouth disease, fowl-plague, contagious pleuro-pneumonia and sheep-pox belong. The virus passes through a Berkefeld filter, but is retained by a porcelain filter. The researches of the investigators named have also furnished us with the following biological relationships of the virus. It is distinguished by great power of resistance to antagonistic efforts. It can successfully bear complete drying and several weeks' exposure to diffused daylight and to sunlight; prolonged influence of a temperature of 10.4° F.; three hours' heating to 140° F.; one hour's heating to 212° F. (boiling-point), when previously dried and sealed up in vacuum tubes; finally many weeks preserva-

tion in glycerine. It is susceptible to the action of carbol, of which a 2 per cent. solution destroys it. The virus of the pigeon-pox suffers such change in even a single transmission through the hen, to which it is communicable without delay, that it can no longer be inoculated on the pigeon ; thus corresponding to the property of the original fowl-pox. The occurrence of a single extended illness produces immunity.

Bacteriology.—Po'owinkin, who has also carried out new researches on pigeon-pox, is of the opinion that the contents of the epithelial cells which were formerly taken for gregarines, coccidia and blastomycetes, indicate degenerative processes. He thinks that he has discovered in the epithelium nodules, and in the blood of the sick animals pathogenic organisms (rods) and has communicated the nodes to healthy pigeons. According to him, the epithelioma contagiosum of birds is morphologically distinguishable from the molluscum contagiosum of human beings. Fowl-pox has nothing to do with that of mammals. Mingazzini thinks that pigeon-pox is caused by the chytridiopsis socius, a parasite which resides in the intestinal epithelium of beetles (" blaps "). Sanfelice thinks he has found a blastomycete which is the cause of pigeon-pox. Jürgens thinks that he infected himself in his experimental inoculations (epithelioma of the size of a cherry-stone on the thumbs with gregarines). Zürn holds the view that from the gregarinous form, micrococci and bacteria are produced by disintegration, which cause the development of the first form ; also that both forms are but distinct developmental stages of one and the same disease. According to Pfeiffer, the diphtheria of fowls is also a single disease, caused by flagellato protozoa. Babes has shown that the flagellato represent quite usual, but not always, pathological parasites of the normal mucous membranes in fowls. On the other hand, the parasitic nature of the gregarines of fowl-diphtheria was previously denied. These form only as the result of changes of the nucleus consequent upon cell-degeneration (Virchow, Kromayer, and others).

Symptoms.—The clinical phenomena of avian gregarinous diphtheritis agrees in all particulars with those of bacterial diphtheritis when these respective complaints affect the mucous membrane of the head. Here, also, the symptoms are essentially those of croupy diphtheritis of the mucous membrane of the mouth, pharynx, air passages of the head, larynx, conjunctiva, etc., with secondary intestinal affections. Gregarines may also give rise to primary and independent enteritis (Zürn).

In gregarinous diphtheritis, the *skin* is much more frequently implicated than in diphtheritis caused by bacilli. The cutaneous affection consists of hypertrophied nodules on the skin, which are known as gregarinous epitheliomata (*epithelioma gregarinosum* of Bollinger, and are identical with the *molluscum contagiosum* of man). Their favourite seats are those

parts of the head that are not covered with feathers: root of the beak, neighbourhood of the nostrils, angles of the mouth, lobes of the ear, parts adjacent to the external auditory meatus, wattles, surface of face, edges of the eyelids, intermaxillary space, and especially the comb. They sometimes spread over the feathered parts of the head, throat and neck, and may occur on the outer surface of the thighs, abdomen, under the wings, and in the vicinity of the cloaca. At first, these epitheliomata appear in the skin, as flat nodules, which soon become prominent, and which vary in size from a poppy seed to a millet seed. Later on, they usually attain the size of a hemp seed. They are of a reddish-grey or yellowish-grey colour ; often show distinctly in their earlier stages of development a peculiar greasy nacreous lustre ; and are rather firm to the touch. Their surface soon becomes covered with a dirty-grey, yellow-brown, or red-brown crust. They are discrete and disseminated in considerable numbers on the erectile tissues, etc. They vary in size according to their age ; and frequently lie rather close to one another, so that the affected parts look as if coarsely granulated ; or they are crowded together in such a manner as to give the appearance of large warts with divisions through them, or of mulberry-like hypertrophies. Even single nodules, to say nothing of groups, may attain the size of a lentil, pea, cherry-stone, broad-bean, or larger object. The older they become, the rougher, and more covered with knobs will be their incrusted surface.

If the edges of the lids of the *eyes* be affected by these tumours, they will become nodular, swollen and closed. The conjunctiva in this case also suffers ; it projects outwards ; becomes catarrhally inflamed ; assumes a yellowish colour at the seat of eruption ; and its surface gets covered with crusts. Purulent conjunctivitis may appear, and the inflammation may spread to the sclerotic and cornea, with keratitis and panophthalmia as the result. If, as sometimes happens with pigeons, the eruption of nodules extends over the whole of the skin of the eyelids and its neighbourhood, the entire eye will become covered with mulberry-like proliferations of various sizes.

Course and Prognosis.—The course of this gregarinous disease is, on the whole, more benign than that of diphtheritis caused by bacteria. The epithelioma of the skin often heals spontaneously, and the nodules dry up and fall off. Gregari-

nous inflammations which are restricted to the mouth, pharynx, and entrance to the larynx, also heal spontaneously at times, or under simple treatment. Other cases, however, run a fatal course, either directly by suffocation (localisation of the process in the larynx and trachea), or from cachexia brought on by extension of the eruption and by increase of the catarrhal inflammation, especially when it involves the intestines. Loss of appetite, depression of spirits, emaciation, ruffled condition of the feathers, etc., are also observed in such instances. Bollinger noticed that fowl died in 4 or 5 weeks after the commencement of the infection, and in 3 or 4 weeks after the first nodular eruption had appeared.

Pathological Anatomy.—The epithelioma nodules of the skin (fowl-pox) are produced by an active epithelial proliferation, with infiltration of the corium and subcutaneous tissue. The large, proliferating epithelial cells are increased to two or three times their normal size; and show, in the most superficial cell-layers, small, homogeneous flakes or a homogeneous mass, which occupies the greatly bloated cell-cavity, and presses the cell-nucleus aside. The changes in the cells of the mucous membrane are similar.

Therapeutics.—The treatment of gregarinous diphtheritis is the same as that of diphtheritis caused by bacilli. We have obtained particularly good results by painting the affected mucous membrane with a 1 to 2 per cent. solution of creolin or carbolic acid in equal parts of glycerine and water (*creolini* 5, *glycerini*, *aquæ destillatæ aa* 100). Pure glycerine alone kills the gregarines by depriving them of water. Glycerine may be also given internally, especially to geese, in doses of a teaspoonful up to a tablespoonful in cases of gregarinous enteritis (Zürn).

Cercomonas.—A third variety of croupous diphtheritic inflammation of the mucous membranes is caused by cercomonas. Rivolta found in the deeper layers of the white-yellowish plaques of fowls that were suffering from croup of the pharynx, œsophagus, and crop, thousands of oval cell-shaped infusoria which possessed the power of exceedingly rapid movement. He gave them the name of *cercomonas gallinarum*, and considered that they were the cause of pseudo-croupy inflammation. Zürn observed cercomonas in pigeons, in which he found that the inflammation caused by them was only slight, and that the yellowish, gelatinous, easily-liquefying coatings of the mucous membranes were only slightly adherent.

II.—Diphtheria of Calves.

Etiology.—Dammann and, more recently, several others have described, under the name of "diphtheria of calves," a diphtheritic inflammation of the mucous membrane of the mouth and pharynx, which seems to occur not only among calves, but also among lambs. Dammann considers that the diphtheria of calves is identical with the diphtheria of man, and that both diseases are produced by one and the same micrococcus. He supposes that calves and their sheds are the hitherto unknown source from which men obtain the diphtheritic poison, and believes that this complaint is contagious, and that it can be transferred by inoculation to lambs, rabbits, and man. It attacks animals particularly during their first few weeks of existence. The period of incubation is very short, but the infectious matter remains virulent for a long time.

Löffler, who investigated diphtheria of calves for the German Imperial Board of Health, believes that the specific cause of the disease is bacilli, and not micrococci, as Dammann assumes. He found on the inner border of the tissue destroyed by this disease, large, long bacilli which formed undulating threads, and which differed entirely from the *bacillus diphtheriæ* of man, both by their form and by their action on the infected parts. Ritter confirmed these statements. More recent researches of Bang, Jensen, Kitt, and others, have shown that Löffler's calves' bacillus is identical with the necrosis bacillus; and that the diphtheria which occurs in various organs of the body in grown-up cows and in horses (intestine, paunch, œsophagus, uterus) is produced by the necrosis bacillus.

Anatomical Conditions.—The principal changes in diphtheria of calves are found in the mucous membrane of the mouth and pharynx. This affection shows itself chiefly on the tongue, hard palate, and inside of the cheeks, as a yellow, croup-like deposit which is partly circumscribed, partly diffuse, and which adheres closely to the underlying tissues. It sometimes attains a thickness of an inch or more. On many places, the croupy membrane entirely destroys its underlying tissues, such as the lamellæ of the bones of the hard palate, and the muscular apparatus of the tongue. These deposits, when examined microscopically, are found to be composed of dense accumulations of micrococci, detritus, threads of fibrine, leucocytes and the long bacilli of Löffler. Similar changes, though to a lesser degree,

are found in the nasal cavity, larynx, trachea, and intestinal canal. Koudelka found in the larynx a greasy, drab-coloured (grey-yellow), burnt-smelling coating, on the removal of which a deep ulcer extending to the cartilage and having an uneven base was disclosed to view. In one case, the interdigital skin of both fore-feet showed an enormous deeply-penetrating diphtheritic infiltration. On *post-mortem* examination, besides the above-mentioned changes, circumscribed, pneumonic foci were found in the lungs with suppuration and purulent pleuritis. The spleen was not enlarged.

Symptoms.—The disease during life closely resembles foot and mouth disease. There is entire loss of appetite, profuse salivation, purulent nasal discharge, and fever. The cheeks and laryngeal region become swollen ; the patient coughs ; and the previously described changes are to be found in the oral cavity. Difficulty of breathing sets in later on, and sometimes diarrhœa. There is great debility, marked inclination to continue recumbent, and rapid emaciation. Death, which is the almost invariable termination, generally takes place in 4 or 5 days ; although the period may be prolonged to 2 or 3 weeks in lung or intestinal cases. Dammann affirms that calves are only susceptible to the disease for the first week of life, and that no cases occur later. Moreover, the course of the diphtheria of calves is more acute and malignant.

Therapeutics.—*Prophylaxis* consists first and foremost in separating the infected animals from the healthy and in a thorough disinfection of the shed. The croupy deposits in the oral cavity should be treated with disinfecting agents, such as carbolic acid, creolin, lysol, salicylic acid, and chlorate of potash, as already stated. If possible the deposits should be removed, and the mouth repeatedly washed out. Dammann recommends the frequent injection into the oral cavity of a half per cent. solution of carbolic acid, or the application, by means of a brush, of a paste made of salicylic acid and water, and the internal administration of salicylic acid.

Diphtheria in Pigs.—Johne observed diphtheria of the upper portions, respectively, of the digestive canal and air passages (glossitis, tonsilitis pharyngitis, and laryngitis follicularis diphtheritica) in several pigs which had been sent together by rail and which had shown great difficulty of breathing and prostration. The most striking changes were found in the tonsils, which were considerably swollen on both sides. The openings of the amygdaloid cavities were pretty well filled with yellowish

crumbling plugs, which could be squeezed out. The section surface appeared yellow, and resembled dry cheese. The neighbouring mucous membrane was stuffed with numerous rounded, grey-yellow [drab-coloured] deposits, which were the necrotic solitary follicular glands, and which were as big as a pea. The bacillus of necrosis was found in the necrotic mucous membrane.

According to Kitt, there occurs sporadically and enzoötically an independent diphtheria of pigs which has no connection with swine fever. He states that it is caused by the bacillus of necrosis, which occurs also in diphtheria of calves. The mucous membrane of the tongue, cheeks, pharynx, and stomach shows yellow-white caseous deposits; and that of the small intestine and colon, diphtheritic necrosis.

Gray, Symes and Bell have made reports on the diphtheria of cats. Diem has observed it in lambs.

PARASITIC STOMATITIS.

Etiology.—This disease, which can be transmitted by man to animals, is a mycosis of the mucous membrane of the mouth and pharynx, and sometimes also of the entrance of the larynx and the upper part of the œsophagus. It is caused by a fungus, which was discovered in 1840 by Berg and Gruby, and which was formerly known as *oïdium albicans.* Gravitz considered this fungus to be identical with *mycoderma vini.* More recent investigations by Plaut have shown that it is a very common mould fungus, *monilia candida*, which grows on fresh cow-dung, rotten wood, and thrives in uncooked cows' milk and on nutritive mediums containing sugar. Plaut has cultivated the fungus and produced the disease in fowls by inoculating pure cultivations. The fungus is found in human beings (sucking babies), calves, foals, and especially in birds. It forms a mycelium, which consists of filaments and roundish, oval or cylindrical shining cells (*gonidia*), and can be stained with Gram's solution.

Pathogenesis.—The settlement of the fungus on the mucous membrane of the oral cavity depends on certain circumstances which have not yet been fully ascertained. Youthfulness, constitutional weakness, gastric disturbances defective cleaning of the mouth, and feeding on milk and on food containing a large proportion of starch, seem to favour the growth of the fungus. Decreased power in chewing and in swallowing appears to assist the adherence of the fungus to the mucous membrane of the mouth, by the prolonged retention in the mouth of food substances which are inclined to form acids. In human beings,

suspicion is especially directed to the mother's milk, a portion of which may be left in the oral cavity of the baby, or may be brought into prolonged contact with the mucous membrane of the mouth by frequent vomiting. Martin observed a case of the fungus being transmitted from a child to a sick fowl.

Anatomical Conditions.—The local affection appears to be restricted in fowls to the mucous membrane of the œsophagus and crop. We notice first of all, on the otherwise quite intact mucous membrane, small white spots, which become enlarged and confluent. They finally form a white, grey, or yellow substance, which is frequently divided by clefts into compartments, and which is of a crumbling or greasy, croup-like consistence. If we remove this membranous deposit, we shall find that the underlying mucous membrane is reddened, or that it has undergone slightly ulcerous changes.

The microscope shows that the described deposits on the mucous membrane are composed of cast-off pavement epithelium, between which the fungus penetrates in the form of fine, decussating filaments, that are plaited into a dense felt. On the ends of these filaments we can recognise the formation of spores, as well as free, spherical or oval, green spores.

Symptoms.—The symptoms in fowl, with the exception of the before-mentioned anatomical changes, are not very characteristic. The affected birds are depressed in spirits, and become emaciated in spite of their possessing a voracious appetite ; the crop is distended ; a sour smell comes from the mouth ; and death ensues with violent convulsions.

Differential Diagnosis.—This stomatitis may very easily be mistaken, as often happens, for other inflammations of the oral mucous membrane ; for croupy diphtheritic stomatitis, especially in fowls ; and for aphthous, pustular, and ulcerous stomatitis in mammals. The essential data for correct differentiation can be furnished only by the microscope.

Therapeutics.—Besides strengthening the patient with good food and by overcoming contingent gastric disturbances, we should be most careful to cleanse the oral cavity and to keep it aseptic. Plaut recommends, in particular, the painting on of a solution of corrosive sublimate (1 to 1,000), as well as the removal of the deposit.

TETANUS (LOCKJAW).

Nature.—Tetanus is an infective disease caused by a specific bacillus. Kitasato, in 1889, isolated the tetanus bacillus which was described in 1884 by Nicolaïer, and obtained a pure cultivation. The tetanus of man, the tetanus of animals, and the tetanus which can be produced experimentally by inoculation with soil, are respectively caused by one and the same pathogenic organism, that is, by the bacillus of Nicolaïer.

Bacteriology.—The tetanus bacillus, in its spore-containing condition, forms, according to Kitasato, rods in the shape of a pin or ladle with a terminal spore 6 to 8 μ long. In its sporeless form, it consists of rods which are just the same length, are straight and rounded off at the ends, and appear singly or in groups from two to four in number. These rods possess a weak automatic power of movement and form long, slightly undulating pseudo-threads. The tetanus bacilli stain well with the customary aniline colours, and also by Gram's method. For the spore containing bacilli, Ziehl's double staining may be used. They are obligatory anærobes and consequently grow only when air is excluded, and better still in hydrogen. Cultivations thrive in slightly alkaline, pepton-containing gelatine, agar-agar, and broth. An addition of grape sugar (2 per cent.) or sulph-indigotate of soda (1 *per mille*) accelerates their growth. The cultivations are characterised by their empyreumatic smell. The single colonies have a certain similarity with cultivations of hay bacillus. A dense centre is surrounded by a fine, uniform wreath of rays. The gelatine slowly liquefies with formation of gas. Tetanus bacilli thrive best in a temperature of from 97° to 100.5° F. Their growth stops at a temperature lower than 57° F. At the temperature of the blood, spores form in 30 hours; and with one of from 68° to 77° F., in not less than a week.

The resistance of tetanus bacilli, especially in the spore form, is very great. Thus, for instance, the action of a 5 per cent. solution of carbolic acid continued for 10 hours, has no effect on the spores (Kitasato). Dried spore-containing tetanus-pus retains its virulence for over 16 months (Kitt). Clams used for castration may remain capable of transmitting the virus of tetanus to horses for eighteen months, even after they have been placed for 5 minutes in boiling water, or in a 4 per cent.

carbolic solution (Nocard). On the other hand, the spores are killed by being exposed to a temperature of 212° F. for from 2 to 5 minutes in a steam sterilizing apparatus. According to Tizzoni and Cattani, mineral and organic acids, even when concentrated to the highest degree, produce no effect whatever on the spores. The spores in the cultivations are, however, killed by a 1 per cent. solution of nitrate of silver in one minute ; by a one-tenth per cent. of the same agent in 5 minutes ; by a 1 per cent. solution of corrosive sublimate in 25 minutes ; by a 5 per cent. solution of creolin in 5 minutes ; by a 1 per cent. solution of permanganate of potash in 10 minutes ; by a 5 per cent. solution of carbolic acid mixed with ½ per cent. of hydrochloric acid, in 25 minutes ; and by a solution of iodine in 6 minutes. Chlorine, chlorinated lime and tar possesses a destructive action on the spores, which, however, resist putrefaction for a long time. Iodine trichloride seems to possess a particularly strong antiseptic effect on the tetanus spores (Behring). The spores are exceedingly resistant to the digestive fluids, especially to the gastric juice. Sormani states that they may grow and increase even in the intestine. On the other hand, animals can be exclusively fed for long periods on pure cultures of tetanus bacilli without injury to their health. According to Fermi, Celli and Colasanti, the poison of tetanus is rendered innocuous by the hydrochloric acid of the gastric-juice. Thalheim holds that infection cannot be carried out in experimental animals on even a diseased stomach and intestine. This coincides with the experiences of meat-inspection : that no ill consequences have hitherto been observed in man after eating the flesh of animals affected with tetanus, which had been condemned to slaughter.

The bacilli of tetanus (pus of wounds, earth) are most easily transferred by inoculation to white mice (scissor-cut wounds on the skin of the croup) ; the experimental animals sicken in 24 hours ; erect the tail in a peculiar manner, and display extension spasm in which the hind legs are stretched out in a straight line (seal-like position). This position continues from 2 to 4 days, and then passes into a fatal paralysis interrupted by convulsive movements.

Pseudo-tetanus bacilli.—A *bacillus pseudotetanicus* has been described by various observers, which occurs in the earth (non-poisonous, or weakened forms of the real bacilli ?).

Pathogenesis.—Like the bacillus of malignant œdema, the

bacillus of tetanus is an ubiquitous bacterium. Numerous inoculation experiments have proved that it occurs everywhere in garden mould and is not restricted to any geographical limit. The inoculation of garden mould from Berlin, Göttingen, Wiesbaden, and other places, in mice, guinea-pigs, and rabbits, was found to produce tetanus of a uniform type. Mould rich in animal manure, especially horse-dung, forms a particularly favourable abode for tetanus bacilli, which are, on that account, constant residents of stables. Sanchez and Veillon found them even in the dung of healthy horses and cattle; Sormani, in the dung of dogs, rabbits, guinea-pigs, and mice; and Molinari in the fæces of pigs, asses, cats, geese and ducks; and from these bacilli produced, by inoculation, tetanus in rabbits. The infection takes place most frequently by the penetration of earth containing bacteria into wounds. Consequently, so-called traumatic tetanus is the ordinary form of the disease. The most frequent causes of tetanus are wounds from splinters of wood or gravel soiled with earth; injuries of the feet by pricks in shoeing and gathered nails; tread with subsequent soiling by dung, excrement or earth; contused wounds which have come into contact with street dust; and operation wounds, especially those of castration, performed without antiseptics. In many localities, and in certain places (infected stables) there are found such quantities of tetanus bacilli that nearly every wound which becomes soiled with earth is followed by tetanus. Tetanus may also be set up by infectious secretions of wounds, either by direct contact, or indirectly, by instruments, to which such secretions may adhere; or by the falling of tetanus spores in the form of dust, on wounds. It has been clearly demonstrated that the dust of hay and cobwebs may be the bearers of tetanus bacilli. Evidently there are many other intermediate bearers. It has not yet been decided with certainty whether infection may take place through the intestines or through the lungs. Experimental tetanus has, however, not yet been developed through the intact mucous membrane of the intestine or the organs of respiration, by way of food or inhalation. On the other hand, an infection has been demonstrated which was conveyed through a respiratory mucous membrane affected with catarrh (Thalmann); and an infection from the diseased intestinal mucous membrane is also probable.

The action of the tetanus bacilli on the animal body is entirely characteristic. The tetanus bacilli are distinguished from most schizomycetes—especially from the septicæmic—

by the fact that they do not pass into the blood and general tissues of the body, but remain at the seat of infection, from which position they poison the body by their metabolic products (toxins). The entrance of the toxins takes place partly along the course of the blood, and partly along the peripheral nerves directly to the spinal cord. According to recent researches (Marburg) the toxin is taken up by the axis-cylinder substance of the peripheral nerves and conveyed to the central nervous system. The settlement of tetanus bacilli in a wound is favoured by simultaneous injurious influences affecting the tissues, such as those due to bruises, foreign bodies, etc.; and also by the simultaneous entrance of other bacteria.

Tetanus is therefore a toxic infective disease, in which a stationary vegetation of bacteria leads to a general intoxication of the body. This circumstance explains the observed fact that inoculation with the blood of animals suffering from tetanus always gives negative results, whilst inoculation with the bacteria-containing pus of the infected part produces positive results. The local colony of tetanus bacilli forms, during vegetation, chemical products (toxins and ptomaines) which have an action similar to that of strychnine, and which poison the body from the focus of infection. Brieger has demonstrated the existence, in tetanus cultures, of four strychnine-like toxins, namely, tetanin, tetanotoxin, spasmotoxin, and a hydrochloric acid toxin. These toxins are rendered ineffective by the gastric juice, intestinal epithelium, by many chemical agents, such as permanganate of potash, and by a temperature of 302° F. (Fermi); according to Kitasato this is brought about at a temperature of only 149° F. They are to be found in very great quantities in the tetanised muscles, in the blood and urine (Courmont), and in the milk (Brieger). Tetanotoxalbumin has lately been mentioned as the most important toxin. This possesses unparalleled poisonous powers; a quarter of a milligramme (= $\frac{1}{200}$th of a single drop) of the solution of the poison suffices to kill a horse of 1,100 lbs. weight (Knorr). Its influence is not only productive of spasm (tetanospasmin), but is also destructive of blood-cells (tetanolysin).

The possibility of the transmission of tetanus from one animal to another, was demonstrated for the first time by the experiments of Carle and Rattoni in 1884. Recently, Kitt, von Behring, Nocard and others have conveyed tetanus by inoculation with pus of wounds and with pure cultures, to horses,

sheep, goats, pigs and dogs with the result of typical tetanic symptoms. Knorr has shown that the various genera of animals differ most extraordinarily in their susceptibility to the infection of tetanus; among the most susceptible of all is the horse and among the least so are fowls.

Tetanus sine tetano.—According to Donitz, two kinds of disease may be produced experimentally by use of the tetanus poison: 1. The usual spastic form, and 2. a variety which leads to a fatal result by marasmus, without tetanus ("tetanus sine tetano"). This latter form does not develop from an overwhelming poisonous power of the tetanolysin (in opposition to the spastic influence of tetanospasmin), but through the fixation of the whole of the tetanus toxins in organs other than the cerebral nervous system (Miyamoto).

Occurrence.—Tetanus occurs in all domestic mammaliæ, but most frequently in horses, asses, and mules; next to them, in the smaller ruminants (sheep and goats); and most rarely in dogs and birds. In the tropics and other hot climates, cases of tetanus including those of man are decidedly more numerous than in colder regions. For instance, Wagenfeld states that tetanus is so frequent in St. Domingo that a gelding costs twice as much as a stallion. In general, however, tetanus is a rare disease. In the Prussian army there is on an average only one case of tetanus in 1,000 sick horses. For instance, 33 cases of tetanus occurred in 1890 among 36,000 sick horses. Within the last 10 years, only 387 horses (40 per annum) were attacked by tetanus in the Prussian army. According to Hering, 1 case of tetanus occurred in 3,000 cases of disease amongst the cavalry horses of Würtemberg. Sometimes the disease breaks out with surprising frequency, indeed epizoötically, among sheep and horses. Röll and others have called attention to the fact that in veterinary hospitals tetanus is often not seen for months, or only very rarely in single instances; but that at other times, 2, 3 or more cases are brought in together. The occurrence of these temporary and local enzoötics is explained by the infectious nature of the disease. Although a direct transmission of tetanus from diseased to healthy animals does not occur, it is nevertheless possible that a horse suffering from tetanus may infect the stable floor for a long time, so that the bacilli of tetanus may get from the floor into accidental wounds of healthy horses. Mestre saw 3 successive cases of tetanus in 3 horses which had occupied the same place in a stable.

Horses of high breed and strong constitution seem more susceptible to tetanus than underbred and less vigorous animals.

A horse seldom gets tetanus twice, though some horses have suffered from tetanus 2 or 3 times within a year. Accordingly, the occurrence of tetanus in the horse conveys no immunity. These facts have also been established by laboratory experiments (Knorr). Among cattle, tetanus most frequently attacks cows and calves; among sheep, lambs; and among goats, bucks. Fowl are supposed to be immune.

Varieties of Tetanus.—Formerly 3 kinds of tetanus were recognised, namely, traumatic, rheumatic, and toxic tetanus. According to recent investigations we must reject the theory of the existence of rheumatic tetanus; for cold, like in other diseases, appears to play only the part of a predisposing agent. Accordingly, we can henceforth recognise but the infective (traumatic) and the toxic forms of tetanus.

Traumatic or infectious tetanus is the ordinary kind. Although experience teaches that any injury may be followed by tetanus; this disease develops by preference in contused and lacerated wounds, gun-shot wounds, punctures in which the foreign body remains, injuries of the nerves, tendons and fascial wounds of the extremities in particular, and especially injuries of the feet of horses, contusions of the paws of dogs, and small and insignificant wounds. In horses, the chief sites of infection are wounds from gathered nails and pricks in shoeing; and to a lesser extent, wounds from tread, corns after having been cut out, suppurating sand-cracks, and especially injuries to the sensitive sole. This predisposition of injuries of the hoof is explained by the theory of the wound becoming infected by dung. Spinola, very long ago, laid stress upon the danger of tetanus from infected wounds. It is probable that injuries of the hoof are much more frequently the cause of tetanus in horses than is usually supposed. Hartmann, for instance, showed that all the nine horses which were infected by tetanus in the course of several years at Dresden were found, on exact examination, to have suffered from hæmorrhagic pricks in shoeing, or injurious pressure from nails driven too " close." The fact is important that tetanus may occur even in wounds already healed and after the formation of a cicatrix (scar tetanus). Among other injuries conducive to tetanus are: saddle and harness galls, fracture of the vertebræ, fracture of the skull, burns, lacerations of the skin during petechial fever, penetration of foreign bodies into carious teeth, clippings, sharp embrocations, etc.

Tetanus may be observed after certain operations, especially

castration. It has been proved by experience that nearly all the buck-goats which are castrated by certain methods, especially the ones by which the scrotum is opened with the knife, die of tetanus. For this reason, castration is now performed by ligaturing the entire scrotum, by including it between clams, or by the subcutaneous ligature of the spermatic cord. Although tetanus in rams may occur independently of castration; in some years it appears enzoötically or even epizoötically as a consequence of that operation. Tetanus is liable to occur after the castration of bull-calves, especially when the scrotum has been ligatured, or when the spermatic cord has been subcutaneously ligatured. In cases of horses, besides castration, other operations—such as inserting a seton, applying a clam in cases of hernia, removal of tumours, docking, nicking, pyro-puncture, so-called frog-cautery (gums), subcutaneous injections, application of cobwebs to bleeding wounds, etc.—may be followed by tetanus, which, in such cases, was more common formerly than at present. The disease occasionally occurs in sheep after inoculation against variola and after the introduction of hellebore root into the nostrils. Since the introduction of antiseptics in operations, cases of tetanus have become rarer than they were; which fact is a further proof of the infectious nature of the disease.

Internal wounds as well as external ones are frequently the cause of tetanus. Thus, cases of tetanus are not very rare in cattle after parturition or after abortion. It appears that the majority of cases of tetanus in cattle are caused during parturition by injuries to the uterus and vagina, difficult births, and putrescent decomposition of the after-birth, etc. Guibert is doubtless correct in saying that in cows, as in women, the obstetrician is sometimes the vehicle by which tetanus is transmitted from a diseased patient to a healthy one. Also, wounds occurring within the buccal and nasal cavities, entrance of foreign bodies (ears of corn) into the air sac, lesions of the intestinal mucous membrane, such as intestinal ulcers (Remond, Hering), may form the starting point of the disease.

In newly-born animals, especially foals and lambs, the open wound at the navel may be the gate by which the infection of tetanus enters the body. In such cases, this disease may occur in lambs enzoötically and epizoötically (*Tetanus neonatorum*, and *tetanus agnorum*), and constitutes a form of what was known as lambs' paralysis. Tetanus is also frequent among older foals.

It has been stated that tetanus can appear without any provable solution of continuity, as for instance by concussion, falls, etc. It is probable that in these cases the infection takes place through an injury which has been overlooked.

Period of Incubation.—The time tetanus takes to appear after an injury varies greatly, being usually a few days. The shortest period on record for traumatic tetanus is 6 hours; the longest, 8 weeks. The time after parturition is, on an average, from 5 to 8 days; although it may vary from a few days to several weeks. According to Ehrhardt, the period of incubation in cows which become affected with tetanus after parturition, ranges from 2 to 3 weeks. In horses the period of incubation after inoculation is 4 or 5 days; and in sheep, 2 to 4 days (Schütz). Surgical experiences show that under certain circumstances a very long period of incubation may occur, inasmuch as tetanus often first appears after the wound has been already fully healed. Sometimes tetanus develops in spite of the wound having been treated antiseptically. The average period of incubation in horses may be taken as varying from 4 to 20 days. In a case observed by Fröhner in the horse, the period of incubation was 40 days; the mild course of this case leads to the conclusion that the grave or mild, rapid or prolonged course of tetanus depends not only on the dose and virulence of the bacilli, but on the shorter or longer duration of the incubation stage.

Toxic tetanus has its cause most frequently in poisoning by strychnine, which is often observed in dogs. Brucin, nicotine, picrotoxine, thebain, caffein, ergotine, and other alkaloids also produce a tetanising effect. By the discovery of tetanotoxalbumins and other tetanising agents, traumatic tetanus appears to be very closely allied to toxic tetanus

Rheumatic tetanus, according to the old view, developed after a rapid cooling of the skin, especially after sweating, by draughts of air, cold showers, remaining in the open during damp and cold nights, etc. It has been stated that the disease generally appears 2 or 3 days after the animal has taken a chill. In other cases there is a longer interval. For instance, Hamon reports that he observed tetanus 8 days after a cold brought on by a shower. In young animals, as in lambs, cold is often regarded as the special cause of tetanus. Blame has often been laid on residence in stables and stalls which were situated on high and open ground; exposure when grazing during rainy weather; and chill after washing the body. Thalmann has conducted experiments on guinea-pigs in association with the pathogenesis of rheumatic tetanus. In that

connection, exposure to cold, in cases of infection from without, had no influence on the course. The inhalation of the bacilli has no injurious effect on the healthy respiratory mucous membrane. On the other hand, infection followed when catarrh was present. According to Thalmann, rheumatic tetanus was apparently produced by tetanus-bacilli which found entrance by the passages of diseased respiratory organs. In a case of rheumatic tetanus in man, Carbonne and Perrero found tetanus bacilli in the bronchial mucus.

General Symptoms of Tetanus.—The tetanic spasm begins usually in the muscles of the neck and head, and spreads from thence to the throat, trunk, and extremities (descending tetanus); but may appear first in the hind-quarters and extend forwards (ascending tetanus). In the former case, the symptoms commence with a stiff, stretched-out carriage of the head, and with spasms of the muscles of mastication. In the latter, with stiffness of the hind limbs and spasms of the tail muscles. The first symptoms are sometimes obscure, and may consequently be overlooked for several days. The disease may, however, break out suddenly with extensive tonic spasms. The spasms frequently begin near the site of the injury. The groups of muscles which become attacked by the spasms are as follows :—

1. *The muscles of mastication.*—The tonic contraction of these muscles is called trismus, or lockjaw. According to the degree of the spasm, the lower jaw remains in more or less close contact with the upper jaw, and consequently prehension of food and mastication are either rendered very difficult, or are entirely stopped.

2. *The other muscles of the head* are spasmodically contracted in different degrees. The spasm of the muscles of the ears causes the ears to be " pricked " and their tips drawn together ; spasm of the recti muscles of the eyes induces retraction of the eyes into the orbit, with protrusion of the nictitating membrane ; spasm of the muscles of the nose produces dilatation of the nostrils ; and contraction of the dilators of the upper lip gives an abnormal shape to the opening of the mouth. The muscles of the tongue, of the swallowing apparatus, of deglutition, and of the larynx, are also attacked by spasm.

3. The contraction of the *extensor muscles of the neck* causes a stiff, stretched-out carriage of the head and " ewe neck." The muscles of the neck become hard and tense to the touch.

4. Spasm of the *extensor muscles of the back* manifests itself by an extremely hard condition of the muscles of the back, loins, and croup. Either the neck is stretched out and the back and

croup are carried horizontally (*orthotonus*), or, what is more frequently the case, the neck is " ewed " (head raised and drawn back) and the vertebral column slightly depressed (*opisthotonus*). A lateral curvature of the cervical vertebræ (*pleurothotonus*) is not so often seen as the previously mentioned conditions : and a convex curvature of the vertebræ (*emprosthotonus*) is rarest of all. The tail, especially in horses, is raised and frequently forms a straight line with the back.

5. Spasm of the *muscles of the limbs* makes the animal assume an attitude in which the fore and hind legs are stiffly stretched out laterally, and respectively, to the front and rear, like those of a trestle. The legs can be bent only with difficulty, or not at all.

6. The contraction of the *muscles which compress the abdomen* gives the animal a tucked-up or " herring-gutted " appearance.

7. The spasm of the *muscles of inspiration* renders respiration difficult.

Besides these spasms, the animals show, in various degrees, heightened sensibility and *increased reflex irritability*, which manifest themselves in excitement, timidity, and exacerbations of the muscular spasms ; although the amount of the irritation may be very slight. Thus, for instance, a loud noise, as the banging of a door, may cause the animal to fall down " all of a heap." On the skin we generally find *sweat*, which, in severe cases and towards death, is very abundant and is diffused over the whole body. In other and especially in slight cases, sweat may be absent. Usually the internal temperature is but little if at all increased. Only in fatal cases is the elevation of temperature constant, though often it is very high (107° or even 109° F.), but only for a short time before death. This high temperature usually continues some time after death and may then rise to 113° F., or even more. In a horse, 24 hours before death, Bayer observed a temperature of 102° F. ; one and a half hours before death, 106° F. ; at the moment of death, 111° F. ; and 50 minutes after death, 113° F. The frequency of the pulse is not increased at the commencement of the affection. In slight cases the pulse may remain normal, which it generally does, with only a trifling increase in frequency, until severe exacerbations set in. In many cases, the rate of the pulse rises only during the last few days, and may then be from 70 to 90 beats *per* minute in the horse. The frequency of the pulse of animals which continue recumbent, is much greater than that of those which keep on their legs. The pulse is often hard

and small, and the wall of the artery spasmodically tense. In many cases the pulse is full, soft, and easily compressible.

Increase in the *rate of breathing* is shown generally at the commencement of the disease, and may become very high when the spasms spread to the muscles of the chest and abdomen. The rate of respiration varies greatly according to the excited condition of the animal. As a rule, it gradually increases in cases which end fatally. It may amount to four times its normal standard, without the frequency of the pulse being increased to any marked extent. When the affection is at its height, the number of respirations in horses rises sometimes to from 80 to 100 a minute. Respiration is usually more or less shallow, on account of the fixed condition of the ribs, and the restriction to the movements of the diaphragm in consequence of the spasm of the muscles which compress the abdomen. We may also notice cyanosis and catarrh of the nasal mucous membrane, coughing, laryngeal, tracheal and bronchial rhonchi; and in fatal cases, symptoms of hyperæmia of the lungs, œdema of the lungs, hypostatic inflammation of the lungs, and often pneumonia caused by foreign bodies.

The first indication of the participation of the *digestive apparatus* in this disease, is afforded by its action on the prehension of ood, which is difficult or even impossible, although the appetite may be unimpaired. Some patients can eat comparatively easily; others, only with much trouble and after great exertion, can manage to consume a portion of their food, which frequently accumulates in the oral cavity, and becomes putrid or falls out. Considerable salivation is present. The slight movements of mastication, especially in horses, are accompanied by squashing sounds and smacking of the lips. The animal can generally partake of sloppy food easier than dry. Regurgitation is very common in consequence of spasm of the pharyngeal muscles. The patients are always fond of playing with the drink placed before them, and make frequent, although very often futile attempts to satisfy their thirst, which increases during the course of the disease.

As a rule, the *peristaltic action of the bowels* is somewhat suppressed, and defecation is always delayed on account of the rigid contraction of the muscles which compress the abdomen. Micturition becomes less frequent and more difficult, even to complete retention of urine, in consequence of which the bladder becomes excessively dilated (spasm of the sphincter vesicæ). The urine has generally a rather high specific gravity and some-

times contains albumen. Its reaction is usually not changed. Frequently occurring and long-continued priapism may be noticed in stallions, and even in geldings.

Consciousness is perfectly preserved with all animals almost till death; although the feeling of terror is usually manifested in a very unmistakable manner. The state of the infected animals arouses in the spectator, and especially in the surgeon who treats them, deep compassion for their helpless condition.

CLINICAL ASPECT OF TETANUS IN PARTICULAR KINDS OF DOMESTIC ANIMALS.

The Horse, in the fully-developed stages of the disease, stands with the head raised and the neck stiffly stretched out, and sometimes "ewed," especially in foals. As we have already said, the ears are stiffly pricked and approach one another, reminding us of the ears of hares; the nostrils are rigid and dilated into the form of trumpets; the eyes are sunken; the nictitating membrane protrudes; the pupils are dilated; the veins of the head are tense and prominent; foam and saliva run from the mouth; the tail is raised, and usually carried a little to one side. To enlarge the area of his base of support, the animal assumes the trestle attitude (spreads his legs out), and does not change it, or, at most, now and then rocks his body to and fro on his stiff legs. The appearance of the face betrays marked anxiety and excitement. Several groups of the muscles, especially the masseter and cervical muscles, are remarkably dry, prominent, sharply defined, as if they had been chiselled out, and evidently in a state of excessive tension. Sweat may be seen on several parts of the body, especially at the base of the ears, sides of the neck and chest, and flanks, which are greatly tucked up. Respiration is difficult and accelerated. If we approach the animal we cannot fail to note the anxiety and terror which it evinces on being touched, and especially if its head be handled. The wings of the nostrils, external muscles of mastication, and muscles of the neck, back, loins, croup and tail, are tense and as hard as a board to the touch. It is difficult to alter the position respectively assumed by the tail and ears, which resume it more or less rapidly on the restraint being removed. All the visible mucous membranes of the head appear congested. The jaws are firmly closed and cannot be forced open. When, for this purpose, the fingers are introduced between the bars of the mouth, considerable quantities of tenacious mucus, or turbid, whey-like fœtid fluid, which is mixed with particles of food, flows from the mouth. On the head being raised, the haw becomes visible to its full extent, so that it frequently covers the greater part of the eyeball. If we attempt to back the animal out of its stall, the movement to the rear is accomplished only with the greatest difficulty or not at all. The horse turns with extreme difficulty without bending the trunk and neck, and only slightly flexes and raises his legs, which he uses like stilts. The gait, which may gradually improve a little, is exceedingly stiff and straddling.

In **cattle** the symptoms of tetanus are frequently modified by the disease being complicated with acute or chronic septic endometritis. The animals assume, with outstretched limbs, the trestle-like, rigid attitude

already described. The head and neck are stretched out ; the ears are stiff and directed backwards, upwards and outwards ; the expression of the face is staring or anxious ; the eyes are deeply retracted into their cavities ; the nictitating membrane, as in the horse, covers the greater part of the eyeball, especially when the head is raised ; the muzzle is sometimes dry and warm ; and the mucous membrane of the mouth is hot, and secretes quantities of tough fœtid mucus. The lips are somewhat drawn back ; the mouth is spasmodically closed ; the lower jaw is firmly fixed to the upper one ; the muscles of mastication are tense and hard as a board ; the tongue possesses little mobility, is hard and sometimes firmly fixed between the jaws ; the muscles of the throat, back and extremities are hard to the touch ; the spinal column is sometimes arched (*emprosthotonus*) ; and the abdominal walls are at times drawn in laterally in a peculiar manner, so that they form a flat surface downwards, from the ends of the transverse processes of the lumbar vertebræ. Great difficulty in breathing is met with only towards the termination of the disease. In consequence of the suppression of the movements of the paunch, tympany of the left flank frequently appears in a few days. Defecation and micturition are delayed. The locomotary movements are stiff, awkward, and often entirely suppressed. The hocks are sometimes rotated outwards, and the tail raised. Reflex irritability is usually less altered than in the horse.

In **sheep** the hind-legs, and, later on, the fore-legs, are stiffly stretched out, so that the animal generally rests immovable as if on four stilts. The tail is rigid, carried straight out, and is frequently deflected somewhat sideways. The neck is stiff and " ewed," and the jaws closed. Later on, the animal drops down and lies with its legs extended straight out, the neck being drawn backwards, on which account respiration is rendered excessively difficult. In other particulars, the symptoms of the disease agree with those in horses and cattle. From existing literature it is difficult to obtain an exact description of tetanus in lambs ; for tetanus in them has frequently been mistaken for other diseases, especially pyæmic polyarthritis. The symptoms of tetanus in **goats** are the same in every respect as those in sheep.

General tetanus appears to occur very rarely in **dogs**. Möller found among 50,000 diseased dogs, only 2 cases of general tetanus. We ourselves failed to discover a single case amongst 70,000. Partial tetanus in the form of trismus seems to occur sometimes in puppies. Recent inoculation experiments prove that dogs are not absolutely immune to tetanus, as was formerly supposed. The symptoms of general tetanus consist in a stiff, stilty gait ; extended attitude of the head ; elevation of the neck ; the ears are stiffly pricked, and sometimes laid back ; staring anxious expression of face ; protrusion of the nictitating membrane ; corrugation of the skin of the forehead ; trismus, which renders the patient incapable of barking ; extension of the muscles of the neck (" ewe neck ") ; stretched-out and stilty attitude of the legs ; orthotonus ; opisthotonus ; erection of the tail ; excessively increased reflex irritability, especially on being suddenly touched. As causes, may be mentioned among others, the pressure of a thorn between the toes (Seitz), and the retention of the ligature, or a docked tail (Grunau). A case of puerperal tetanus in the dog has been described by Duschanek.

In **pigs** the symptoms of tetanus are essentially the same as in other animals. The tonic spasm usually begins in the muscles of mastication and spreads rapidly, as a rule, over the entire body (trismus, orthotonus and emprosthotonus).

In **birds**, Dreymann has described one case, namely, that of a turkey. The gait was stiff, the head and neck were extended, and the entire muscular system was stiff and hard. The beak could be opened only with difficulty, and finally became firmly closed. The wings lay close to the body. The membrana nictitans was protruded, and dyspnœa was present. In opposition to Kitasato, who has affirmed that fowls are completely immune to tetanus, Courmont and Doyen, as well as Knorr, have successfully inoculated fowls with tetanus-bacilli. According to Knorr, however, fowls are 200,000 times less susceptible than the horse. These birds sickened only when the amount of the dose administered had reached a quantity capable of causing the death of 1,000 horses.

Anatomical Conditions.—The results of a *post-mortem* examination of a case of tetanus are usually negative. Similar to what is the case in narcotic poisoning, there are no characteristic anatomical changes. Any changes that may be found are only secondary, or are set up by accidental complications. Thus we may sometimes find in the spinal cord, hæmorrhages, softening, accumulation of serum, hyperæmia, œdema, etc., which changes, however, may be entirely absent, or only secondary, and are frequently traumatic. We may find in the course of the nerves which proceed from the wounded parts, in some places, a pink injection of the nerve sheaths, very minute hæmorrhages, and swelling or softening of the nerve fasciculi (*neuritis ascendens*). On account of impeded oxygenation, the blood is usually of a dark-red, tarry and greasy appearance, is greatly discoloured, and is imperfectly coagulated. It contains free hæmoglobin, and is inclined to escape from its vessels and to become absorbed into the surrounding tissues. We may observe numerous extensive ecchymoses, and large sanious and gelatinous extraversations, especially under the serous and mucous membranes. The lungs are generally hyperæmic or œdematous, and hæmorrhagic infarcts, splenisations, hypostatic pneumonia, lobular, gangrenous pneumonia, and vesicular and interstitial emphysema, may also be present in some parts. In the heart we find epicardial and endocardial hæmorrhages, particularly along the furrows of the blood-vessels and over the papillary muscles, and fatty degeneration of the myocardium. Rigor mortis, which is generally highly developed, appears rapidly in the muscles, which may be in a normal

condition, or may be of a dull, dark-red colour, and infiltrated with hæmorrhages. On other occasions it is bluish-brown, or yellow-brown, and soft as if boiled. In the muscles, we can sometimes find, by the microscope, a granular cloudy swelling, with loss of transverse striation and splitting up of the muscular fibres in layers. The liver is usually enlarged and is frequently of a yellow or yellow-grey colour (fatty degeneration and infiltration). The spleen is swollen, gorged with blood, and flabby. The bladder is usually considerably distended by the accumulated urine, and its mucous membrane is often infiltrated with hæmorrhages. Ecchymoses are not uncommonly met with on the mucous membrane of the intestines.

Prognosis.—The prognosis of tetanus is usually very unfavourable The mortality amounts in sheep and pigs almost to 100 per cent.; in horses from 75 to 85 per cent.; and in cattle from 70 to 80 per cent. In dogs it is comparatively small. Out of 387 horses which were treated for tetanus during the years 1887 to 1897 in the Prussian army, 317 died. In newly-born animals the disease is usually fatal, especially in lambs. In cattle, the prognosis of a case of tetanus following parturition is less hopeful than of one of ordinary tetanus. The chief unfavourable symptoms are: early and excessive trismus (horses in which trismus is not followed by general tetanus mostly recover); rapid spreading of the spasms over the entire muscular system; excessive dyspnœa; rapid and considerable increase in the rate of the pulse and in the height of the temperature; falling down; profuse sweating, etc. The fact of the disease running a protracted course is favourable. With horses we may hope for recovery when the patient lives beyond the fifteenth day of the disease.

Schindelka observed 4 cases of tetanus in which a complication with influenza had a peculiarly favourable influence. In all 4 cases the muscular rigidity abated simultaneously with the commencement of the first feverish symptoms, and recovery followed. Bassi states that a similar effect was produced by parturition in a mare.

Course.—The course of tetanus is variable. In horses, it is frequently very brief, and then ends fatally in 1, 2, or 3 days. Recovery in rapidly developed cases has never been observed. The animal exhibits at an early period of the disease, excessive trismus, spasms over the whole body,

use the catheter in females; and enemas with the smaller animals.

As regards medicinal remedies, we may employ narcotics, at least, tentatively, as for instance, enemas of chloral hydrate of 1¾–3½ oz. nearly, for horses in a thin decoction; subcutaneous injections of morphia (6-9 grs. for horses); inhalations of chloroform; injections of ether into the rectum (⅞-1¾ oz. *pro dosi*). Medicines should not be administered *per os* on account of the danger of trismus being set up, and on the liability of the drench or ball going the "wrong way."

In cases of traumatic tetanus, it is of the utmost importance to apply as soon as possible appropriate treatment to the wound by disinfecting it, removing from it penetrating foreign bodies, excising or cauterising the infected part, or even performing amputation. Animals can be undoubtedly saved by this local treatment, if it be early employed and if it be practicable. We must, however, remember that an exact examination and special treatment of the wounds in the larger animals may be attended by considerable difficulty. With injuries of the feet of horses, it is frequently nearly impossible to raise the leg off the ground without incurring the risk of causing the animal to fall down. If, in such a case, the condition of the wound urgently requires local treatment, nothing is left to us but to place the horse under the influence of chloroform, by the aid of which we may perform the necessary operation. We have not sufficient *data* at present to pass a final judgment on the value of neurotomy in traumatic tetanus. Rosco states that plantar neurotomy was followed by rapid improvement and cure in a case of tetanus caused by a gathered nail.

As *prophylactic treatment*, we recommend the careful disinfection of the place of residence of animals suffering from tetanus. Clinical experience has taught us that the stalls and loose boxes of infected horses continue, for several years, to be dangerous centres of infection (Hartenstein). How far the tetanus antitoxin may be regarded as of practical value before operations, remains undecided (Nocard, Labat).

Immunity against Tetanus.—Behring and Kitasato have experimentally proved that animals may be rendered immune to tetanus by successive attenuated pure cultivations of tetanus bacilli which have been suitably treated with iodine trichloride. This artificially-produced immunity depends on the fact that cell-free blood serum of these animals possesses the power to render inert the toxin produced by the tetanus bacilli. The system of the animal which has been protected by inocula-

tion, consequently contains an effective chemical substance, namely, an antitoxin, on the presence of which, in the blood, the power of immunity depends. Inoculations with the blood serum of animals which have thus been rendered immune to tetanus, are supposed to confer on other animals immunity against subsequent infection with tetanus toxin, or infection with living tetanus bacilli. Behring made these experiments with small animals, such as rabbits, guinea-pigs, and mice. Schütz, Koch, Behring, and Kitasato carried out this method of obtaining immunity in horses and sheep. The animals experimented upon were repeatedly injected subcutaneously with different quantities of cultivations of tetanus bacilli, which had been mixed with iodine trichloride and had thus become weakened. The proportion of iodine trichloride, which at first was from half to one per cent., was gradually reduced to a quarter and then to an eighth per cent., so that the cultivations used for the immunising injections were of increasing virulence. This treatment, which was continued for about a month with intervals of several days, rendered the experimental animals immune to even fully lethal cultures of tetanus bacilli, which proved fatal to the unprotected control animals. From these experiments Schütz arrives at the following conclusions :—" Horses and cattle can by Behring's method be protected not only against an infection with living tetanus bacilli, but also against the injurious effects of the virulent substances which are formed by the tetanus bacilli in cultivations or in the animal body. The curative serum (*tetanus-antitoxin*) introduced in 1896 by Behring, and recommended as a curative agent in the treatment of tetanus, had at first a favourable reception from veterinary practitioners, but was soon rejected. The preparations, more especially those which have been placed on the market during the last years, have, in many cases, proved quite inefficient as remedies against tetanus in the horse. For this reason, and also owing to its high price, tetanus-antitoxin is scarcely used at present as a remedy by veterinary surgeons. Nocard recommends it as a protective agent before surgical operations.

In 1898 Wassermann showed that the brain substance possesses an antitoxic power (tetanus-binding power of the ganglion cells, not of the filtrates). In the years 1901 and 1902, there were researches carried out in this connection with brain-emulsion in the cases of 20 horses affected with tetanus at Vienna (Schindelka, Fiebiger), after the following manner : The brain of freshly slaughtered lambs was thoroughly ground with physiological salt solution, filtered, and the emulsion, rendered as completely sterile as possible, was injected subcutaneously on both sides of the neck of the diseased horse, in quantity of about a pint. In most instances abscesses formed at the seat of the injection, and proved very troublesome. Nevertheless, the brain emulsion appeared to exert a favourable action. Of 20 tetanic horses which were injected, only 8 died, *i.e.*, 52 per cent., against 80 per cent. as formerly. The value of the injection cannot, however, be definitely affirmed ; as after several cures a large number of horses died successively, in spite of the administration of the emulsion. The emulsion treatment appears to render inert the free toxins circulating in the body ; but not to remove or to paralyse the poison located in the ganglion cells of the central organs, nor avert the consequences of the toxic action already existing. The further researches which were carried out with brain emulsion, both on horses already affected with tetanus, and as a prophylactic in healthy ones,

by Ludewig and Landi, have had but partial results. More exhaustive researches must, accordingly, be awaited.

The *neural* (direct injection into the nerve trunk) has been recently recommended with the view of neutralising the toxins ascending from the periphery to the centre, and also the *intracerebral* injection of antitoxin, as the surest methods of combating the disease. Villar advocates the latter method for horses. The injection of the canine serum produces no result (Tennert). In two cases of horses suffering from tetanus the effect of extreme fright (by a shot fired in the stable) seemed to have a curative influence (Kissuth).

GLANDERS (*Equinia, Malleus*).

Glanders in horses—Glanders in cats and beasts of prey—Glanders in man—Lymphangitis epizootica.

History.—Glanders (glanders and farcy) is one of the oldest known diseases of horses. Aristotle and Hippocrates were acquainted with farcy, and glanders of the donkey. The Roman authors, Apsyrtus and Vegetius, have described glanders, and Vegetius recognised several kinds of glanders ("*malleus*"), especially that of the nose and skin ("*malleus humidus et farciminosus*"). In the Middle Ages, glanders was considered, by the German laws, to be a legal unsoundness (breach of warranty). The names used for it were "Hauptmönigkeit," "Haubetisch," "Mordisch," "profluvium atticum." The infectiousness of glanders was recognised as early as the seventeenth century. Solleysel (1664) supposed that it could be transmitted by the air. Van Helmont (1682) considered it to be identical with human syphilis. Even in 1734, Gaspard Saunier gave precise directions about disinfecting stables. Garsault (1741) and Bourgelat (1764) recommended immediate slaughter of horses suffering from glanders and segregation of suspected animals. The identity of glanders and farcy was clearly recognised at that time. At the end of the eighteenth century, Abildgaard and Viborg in Denmark proved by a great number of experiments that glanders could be transmitted from one horse to another. These investigators demonstrated, at this early period, that the virus of glanders was fixed; that it could be transmitted by the respiratory air; that the transfusion of the blood of horses suffering from glanders is far less efficacious for transmitting the disease than inoculations of

secretions or of pus; and that the virus loses its power of infection if dried or heated to 113° F. Similar experiments were made in England by Coleman and Delabère-Blaine, who recognised that the nasal discharge was the principal vehicle of the virus.

On the other hand, the theory of the contagiousness of glanders was much doubted at the beginning of last century. Especially in France, the view taken by the Alfort Veterinary College, that glanders might arise spontaneously from an attack of strangles, was far more widely accepted than the doctrine of its contagiousness, which was stoutly supported by the Veterinary College of Lyons; the consequence being, that precautionary regulations against glanders were abolished, with the result that the spread of the disease increased to an extraordinary extent in France. At that time, glanders was looked upon as tuberculosis (Dupuy), or as simple pyæmia (Vatel and Bouley). It was only when Rayer (1837) had proved the transmissibility of glanders to man, and when Chauveau (1868) had shown that the virus was contained chiefly in the firm component parts of the infective material, that the fact of the contagiousness of the disease was again accepted.

The theory of the spontaneous origin of glanders was accepted by every one in Germany towards the middle of last century. It was believed that glanders could be produced by the injection of pus; and that strangles could turn to glanders (Hering and Funke). Glanders was looked upon, respectively, as a tubercular disease, scrofula (Haubner), pyæmia, diphtheritis, general dyscrasia, and cachexia. Virchow was the first to declare that the nodules of glanders were independent anatomical formations, which he placed under the heading of granulation tumours. Gerlach was the great advocate for the exclusively contagious origin of glanders. He threw much light on the clinical and diagnostic aspects of the disease. We are indebted to Leisering for the first exact anatomical description of glanders.

The first bacteriological researches were made in 1868 by Zürn and Hallier, who found a fungus which they believed to be the cause of glanders, and to be identical with that of human syphilis. In the same year, Christot and Kiener thought they had discovered the micro-organism of glanders. Bouchard, Captain, and Charrin, in 1881–82, sought to isolate the virus of glanders. It was not, however, until the year 1882 that Löffler and Schütz succeeded in finding the true bacillus of glanders, in cultivating it, and in transmitting it to other animals. Their researches have furnished the positive proof that glanders is a

specific infectious disease, produced exclusively by the bacillus mallei.

Bacteriology.—According to Löffler and Schütz, the bacilli of glanders are rods, the length of which is from 1 to 3 μ, or from one-third to two-thirds the diameter of a leucocyte. They are from 5 to 8 times as long as they are thick, are straight or slightly curved, and have rounded ends. They are somewhat thicker and shorter than tubercular bacilli, and are generally found in couples connected together longitudinally. They stain in solutions of methylene blue. The cultures on gelatine appear on the surface of the gelatine, as small, limpid, yellow, translucent drops, and assume, later on, a milky-white colour. The potato cultures, which are very characteristic, form after the third day a uniform, amber-coloured, translucent covering, which assumes in about a week a reddish tinge similar to that of sub-oxide of copper. A greenish hue appears in the neighbourhood of the cultures. The bacillus grows best on solid blood serum of horses and sheep; on neutral broth made of the flesh of horses, cattle, fowls, and human beings; and on slices of potato. The temperature limits of the growth of the fungus are 68° F. and 113° F.—the maximum being from 98° to 100° F. Consequently, summer heat favours its growth, which is impeded or destroyed by temperatures under 68° F. and over 113° F. For this reason it is impossible that an ectogenous development of the fungus can take place. Löffler failed to obtain cultures on infusions of hay, straw, or horse-dung. Glanders appears therefore to be a purely contagious, and not a miasmatic or miasmatic-contagious affection. As a rule, complete desiccation of the bacilli of glanders destroys their virulence in about a week. According to the experiments of Löffler, a period of 3 months is the longest time the dried bacilli retain their activity. Cadéac and Malet state that the bacilli can be killed only by gradual drying; that they resist putrefaction from 14 to 24 days; and that, when mixed with water, they continue virulent from 15 to 20 days. Bacilli which are not dried, cannot live outside an animal's body for longer than 4 months. Löffler therefore considers that 4 months is the maximum period for the infectious material to retain its virulence, and that the published reports about stables remaining infectious for many months, and even years, are erroneous. Löffler regards the formation of spores to be very doubtful on account of the slight power of resistance possessed by the bacilli. Rosenthal, however, treated

glanders bacilli taken from old potato cultures after Neisser's method of staining spores, and obtained exactly similar forms as with the spore-containing bacilli of anthrax. We must therefore admit the possibility of spore-formation. Recently, the bacillus of glanders has been referred by Conradi and others to the actinomyces group.

The power of resisting disinfectants possessed by the glanders bacilli is but slight. Sunlight rapidly destroys them. They are also killed by a momentary boiling ; an exposure to a temperature of 131° F. kills them in 10 minutes, or 176° F. in 5 minutes ; and they die if left for 5 minutes in a 3 to 5 per cent. solution of carbolic acid or creolin, or for 2 minutes in a watery solution of chlorine, a 1 per cent. solution of permanganate of potash, or in a one-fiftieth per cent. solution of corrosive sublimate, etc. The experiments of others show that the bacilli are not always destroyed by an exposure of half-an-hour to a temperature of 212° F. (Bromberg). For practical purposes we may say that a 1 to 1,000 solution of corrosive sublimate, or a 3 per cent. solution of creolin or carbolic acid, is sufficient for disinfection. Iodoform considerably impedes the development of the bacilli outside the animal body, so that their infectiousness is lost when they have been for some time under its influence. Inside the animal body, iodoform impedes but little the development or the dissemination of the bacilli. According to the experiments of Gamaleïa, the virulence of the bacilli of glanders may be increased by passing it through the body of the earless marmot [*spermophilus citillus*], or rabbit. Although the digestive fluids weaken fresh cultures of the bacilli of glanders, they do not kill them (Mozarski).

Occurrence.—Glanders is essentially an equine disease and affects donkeys, mules and jennets, as well as horses. It is transmitted from the horse to many other animals, including man, either in the natural course of events, or by experimental infection. The order of comparative susceptibility to the virus of glanders possessed by certain animals is somewhat as follows :—Field-mice and guinea-pigs ; the cat tribe, including lions and tigers ; dogs, goats, rabbits, and sheep. Pigs and pigeons are scarcely at all susceptible. Cattle seem to be quite immune. In fact, there has never been a single well-authenticated case of glanders observed in them. The long-established practice of isolating glandered horses in cowsheds has never produced any bad effect on the cattle residing in these abodes.

House-mice, white mice, rats, domestic fowl, and linnets are immune.

There is a great difference in the individual susceptibility of horses to the virus of glanders, which some readily take, while others may remain for months and even years in a stable with glandered animals without becoming infected. Glanders, like tuberculosis, has certain predisposing factors, the chief of which are: over-exertion, too little food, faulty stable management, defective ventilation, chill, and disease. The spread of glanders is therefore greatest during times of war.

Although glanders is far less frequent at present among horses than formerly, thanks to veterinary police laws, it still continues to be the most dangerous equine disease. It occurs in all latitudes and in all climates. According to the yearly reports of the State sanitary officials on the distribution of epidemic diseases of animals in Germany, the specially affected districts are the eastern borders of the provinces of West Prussia, Posen and Silesia—especially the parts adjacent to Russia. The principal foci are: Dantzic, Bromberg, Königsberg, Marienwerder, Gumbinnen, Inowrazlaw, Posen, Oppeln, Berlin, Potsdam, Liegnitz. In Southern Germany, the destructive foci are especially in Upper Bavaria, Würtemberg and Lothringia.

Statistics of the Disease.—1. In German territory there were officially notified during the years 1886–1902, nearly 13,000 horses (12,780), as suffering from glanders, or nearly 1,000 cases per annum. The respective years show the following numbers:—1,220 (1886); 1,228 (1887); 1,182 (1888); 1,337 (1889); 866 (1890); 981 (1891); 823 (1892); 564 (1893); 516 (1894); 590 (1895); 505 (1896); 338 (1897); 371 (1898); 461 (1899); 748 (1900), 699 (1901), 361 (1902). A steady and very considerable decrease is thus shown. On an average throughout the German Empire 1-2 cases of glanders occur in every 10,000 horses (= 0.01-0.02 per cent.).

2. Felisch computes that from 1876 to 1886, 20,566 horses in Prussia died of glanders or were killed on account of having glanders, and gives the following figures for the respective years:—2,740 (1877); 2,848 (1878); 1,994 (1879); 2,182 (1880); 2,357 (1881); 2,297 (1882); 1,568 (1883); 1,879 (1884); 1,525 (1885); and 1,176 (1886). According to this, the losses diminished considerably more than one-half during these ten years. The decrease began with the introduction of the Imperial laws for the regulation of epizoötics. The principal losses were in Posen (0.22 per cent.), West Prussia (0.15 per cent.), and in Silesia (0.13 per cent.). The disease was most rife in the districts of Oppeln, Bromberg, Posen, Marienwerder, and Dantzic (Russian and Polish-Russian frontier). Draught animals formed 22 per cent. of the glandered horses. There were 20 known cases (3 of veterinary surgeons) of the disease having been transmitted to man. The total sum paid as indemnity amounted

to £212,500. In Saxony, the great increase of glanders in 1900 was chiefly due to the fact that a touring circus infected 17 localities within the space of two years.

3. According to Röll, there were 3,317 horses officially reported as being glandered in Austria during the years 1877 to 1887; or 3 per 1,000 (0.3 per cent.) of the total number of horses. In Hungary, there were nominally affected during the years 1897-8, a total of 2,459; in 1899 1900, a total of 1,700.

4. In France about as many horses as in Germany (5,623) became infected from 1880 to 1890.

5. The number in Great Britain during the same period amounted to 8,000. In 1890 there were 782 cases of glanders in London alone. In 1901, 1,828 cases of glanders were recorded in London alone (in London and its neighbourhood, glanders always prevails as an enzootic disease, while in the counties it occurs only sporadically).

6. According to a table compiled by Krabbe for a period from 1857 to 1873, there were yearly among 100,000 horses in Norway 6 cases of glanders; in Denmark, 8·5; Great Britain, 14; Würtemberg, 77; Prussia, 78; Servia, 95; Belgium, 138; France, 1,130; and in Algiers, 1,548. It would be difficult to decide whether this increase of glanders from north to south, as Löffler assumes, is due to greater warmth of climate (the bacillus of glanders, as we have already said, thrives only at a temperature of over 68° F.), or to insufficient veterinary police regulations in the countries in question.

7. During the 10 years from 1877 to 1887, there were 4 cases of glanders each year among 100,000 horses in Sweden (Lindqvist).

8. Woronzow estimates the number of glandered horses in European Russia at 90,000, that is, 4 *pro mille*.

Pathogenesis.—The transmission of glanders from an infected animal may take place directly or indirectly through extremely different intermediate bearers, such as harness, clothing, pails, stable tools, fodder, litter, grooms, and other agents. The respective discharges from the cutaneous ulcers and from the nostrils possess the highest degree of infectiousness. The bacilli of glanders may occur in any organ as well as in the blood, which does not always contain them, except when the infection is general throughout the system. Cadéac, Malet, and others frequently obtained positive results from inoculation experiments with the blood of glandered horses. The bacillus of glanders cannot multiply outside the animal body (a pure contagious disease; so-called obligatory parasite).

The *respiratory organs* are most frequently (in nine-tenths of all cases) the gate of entrance for the virus. The bacilli, like those of tuberculosis, probably reach the nasal mucous membrane by being inhaled along with the air in the form of dry dust; by the reciprocal smelling of neighbouring animals; and by deep respiratory movements, as for instance, during

severe harness work. We may here point out that the presence of cartarrh predisposes the respiratory mucous membrane to the penetration of the bacilli. According to the experiments of Renault and others, it seems to be very doubtful that the bacilli of glanders can be directly exhaled into the atmosphere and thus brought into contact with the air passages of healthy animals by inspiration. It is much more reasonable to assume that the transmission of the bacilli takes place by the bacilli-laden nasal discharge flowing out or being blown out, and by its becoming dried and then inhaled in the form of dust. Experiments to transmit the disease by the direct inhalation of the respiratory air of glandered horses have always been without result. According to Galtier, infection by inhalation can take place only when the inspired dust contains fresh virus; the *contagium* of glanders is pretty rapidly destroyed by drying. It still remains an open question whether or not the nasal mucous membrane must be morbidly affected before it can successfully receive the infection of glanders; although an affirmative answer has apparently been given by the experiments of Cadéac and Malet. It is probable that the bacilli of glanders, like those of tuberculosis, can penetrate through the uninjured mucous membrane. They may also be introduced along with the inspired air into the lungs, where they then lead to the development of very minute bronchial-pneumonic foci. Glanders in the lungs, like glanders of the air passages of the head, may occur primarily. In fact it frequently happens that the lungs alone are affected, or that they show the oldest changes. For these reasons most authors believe in the possibility of a primary affection of the lungs in glanders (Leisering, Gerlach, Bollinger, Roloff, Pflug, Rabs, Röll, Csokor, Löffler, Peters, and others. On the other hand, according to Schütz, primary glanders of the lung is very rare in horses.

The *skin* is a second gate of entrance for the bacilli of glanders; farcy, which was formerly regarded as a separate disease, being nothing else than glanders of the skin. A primary infection of the skin is, as a rule, possible only when the skin has been previously injured. Cases of primary glanders of the skin are consequently uncommon. The skin more frequently becomes infected secondarily as a result of general glanders set up by emboli. Contrary to the case reported by Babes, Nocard failed to make the bacilli of glanders penetrate the uninjured skin of donkeys and guinea-pigs, by the inunction of a bacilli-containing ointment. If infection takes place after

such an application, we must assume either the presence of an abrasion of the skin, or that the continuity of the skin has in some manner been injuriously affected by the friction employed. According to the more recent researches of Nocard and Fritsche, animals can also be infected by bringing the glanders bacilli into contact with the uninjured—even the unshaven—skin. Cases of primary glanders of the eyes (glanderous keratitis) have been occasionally observed (Richter, Regenbogen, and others). According to the researches of Galtier, infection takes place also through the intact ocular mucous membrane.

In horses, the virus of glanders penetrates the body least frequently through the *digestive canal.** This fact has been proved by the generally negative results which Viborg, Gerlach, Liautard, Cadéac, Malet and others, obtained from their experiments of feeding horses on glanderous matter; although a few of these experiments gave positive results. Renault succeeded in infecting 6 horses out of 9 by feeding them on similar material. Nocard and Sadowski were repeatedly successful in transmitting glanders to horses and asses by feeding. The rare cases of the infection of sucking foals may, omitting the supposition of infection through milk, be ascribed to infection through the inspired air. On the other hand, dogs, cats, and wild animals (lions, tigers, panthers, and bears) become infected, comparatively frequently, by feeding on glandered flesh, in which cases it is possible that small injuries of the mucous membrane of the mouth may form the starting point of the invasion of the bacilli. No case of infection by the eating of glandered meat has yet been observed in man; although many glandered horses have been eaten by human beings, for instance, during the siege of Paris.

Whether cases of transmission observed in coition are those of infection of the genital mucous membrane or of infection by the inspired air, cannot always be decided. Only in a few cases have glanderous ulcers been found on the vaginal mucous membrane of mares. There is, however, no doubt that the bacilli of glanders, like those of tuberculosis, may pass from the mother to the fœtus.

Glanders may spread from a local centre in the same manner

* For arguments in favour of the ingestion theory of the transmission of glanders, see the remarks of Nocard (*Journal of Comparative Pathology*, Dec. 1897) and Hunting (*Veterinary Record*, 8th Jan. 1898) on that subject. McFadyean (*Journal of Comparative Pathology*, Dec. 1897) takes the same view as Friedberger and Fröhner.—Tr.

as tuberculosis. It proceeds at first by way of the nearest lymphatics. In the last stage of a chronic course, the process may be restricted for a long time to the lymph glands. By means of the blood-vessels, the bacilli may reach distant organs such as the lungs and skin. A focus of glanders in the lungs, by invading a bronchus, and by thus contaminating the expiratory air, may set up a secondary infection of the mucous membrane of the air passages of the head, larynx, and bronchi. In acute glanders, the bacilli are rapidly absorbed into the blood and thus set up a general diseased condition. We have no exact information about the part played by urine, saliva and sweat, as vehicles of the contagium.

Varieties of Glanders.—According as glanders is situated in the internal or external organs, we distinguish glanders of the nostrils and glanders of the lungs from glanders of the skin. In former times, the term glanders was restricted to glanders of the nostrils and lungs ; that of farcy being reserved for glanders of the skin. When the animal was affected internally and externally at the same time, he was said to be glandered and farcied.

According to its progress, we have chronic and acute glanders ; the former being the ordinary, the latter the rarer kind. Acute glanders rapidly ends in death and never becomes chronic ; though chronic glanders usually becomes acute. There are many causes why glanders should be sometimes acute and sometimes chronic. The chronic form assumes an acute type if the disease becomes general, that is, if the blood gets infected with great numbers of bacilli. In the same way, ordinary chronic tuberculosis turns into acute miliary tuberculosis. As a rule, chronic glanders ends fatally in consequence of its producing a general infection of the body, that is to say, general acute glanders. Inoculated glanders, and primary glanders of the skin due to wounds, frequently manifest themselves in the acute form. The same thing occurs in those cases of glanders in which a serous infection of the wound takes place, or in which the infected animal suffers from the beginning of the attack from fever. The quantity and virulence of the absorbed bacilli and individual idiosyncrasy also seem to influence the progress of glanders.

Anatomical Conditions in Chronic Glanders.—The anatomical changes in chronic glanders are specific inflammatory

processes which are accompanied by suppuration, ulceration, granulation, and cicatrisation. Their most frequent seats are on the respiratory mucous membrane, in the lungs, lymph glands, skin, and subcutis, Other organs are not so often invaded.

1. *The respiratory mucous membrane* is the usual seat of glanders (glanders of the nostrils). It occurs in two forms: as circumscribed nodular glanders, with the formation of ulcers and cicatrices; and as diffuse or infiltrated glanders.

Nodular glanders, which is the ordinary kind, is most frequently situated on the nasal mucous membrane, and is then chiefly found in the upper portion of the nasal cavity, namely, on the nasal septum, and in the cavities of the turbinate bones. The affection commences with the appearance of nodules, which vary in size from a grain of sand to a millet seed, and which are of a glassy, translucent, gelatinous condition, of a roundish or oval shape, and of a dirty-grey or greyish-red colour. These nodules project somewhat above the surface of the mucous membrane, are surrounded by a red ring, and may attain to the size of a pea, as a maximum. Some of them are isolated, and others are arranged in groups. Microscopically they consist of a large number of lymphoid cells, which break down in the centre, with the bacilli lying between them. In consequence of the fatty and purulent disintegration, which sets in very rapidly in the centre, the nodules become yellow and change into ulcers after the purulent breaking down of their summits. These ulcers are sometimes superficial (lenticular), sometimes deep, crateriform, surrounded by a hard, prominent edge, and frequently, on becoming confluent, form large ulcers which have irregularly serrated and eroded edges and a lardaceous base. They are sometimes covered with a brownish crust. The ulcers may increase in area or in depth, and may even involve the underlying cartilage or bone, thus causing perforations of the septum nasi, distensions of the maxillary bones, exostoses on the turbinate bones, etc. The shallow lenticular ulcers may heal without leaving any visible changes; but the deeper ones, after granulating, leave a radiating, star-shaped, or ice-flower-shaped* cicatrix, which is either smooth or horny, and which, according to the form of the ulcer, may be of an irregular oblong shape. The nasal septum, or the alæ of the nostrils, are frequently covered with these scars. These ulcers and cicatrices are also found in the maxillary and frontal sinuses, in the guttural pouches, and in the Eustachian tubes,

* The ice-flower is the *mesembrianthemum crystallinum*. -Tr.

where they may produce pachydermatous thickening of the mucous membrane with accumulations of a muco-purulent secretion. They are also frequently met with in the larynx, and especially in the region of the lower vocal chords, from which the suppurating process may extend to the arytenoid cartilage and cause its partial necrosis; in the trachea, particularly on the anterior surface, on which numerous long, oval ulcers of the size of a coffee-bean up to that of a hazel nut, or long, pointed, serrated scars are frequently situated; and in the bronchi. In addition to these ulcers, a catarrhal inflammation of the mucous membrane is always present.

Diffuse or infiltrated glanders manifests itself as a diffuse catarrh of the mucous membrane of the nasal cavity, and neighbouring cavities, with superficial ulceration, thrombosis of the veins, inflammatory infiltration of the submucosa, considerable thickening of the mucous membrane, and the formation of a peculiar, radiating cicatrix.

2. In the *lungs*, both the nodular and infiltrated forms are found.

In *nodular glanders*, we find in the lungs nodules* varying in size from a grain of millet up to that of a pea. They are grey by transmitted light, glassy and pearl-grey by reflected light, and are surrounded by a hæmorrhagic ring. The centre of the nodules shows a pale yellow point in consequence of caseation and disintegration of the innermost cells. These nodules are of different sizes, of varying numbers, and of different ages. Along with new ones, we find caseous, calcareous (the calcification of the nodules of glanders is disputed by some), and encapsuled nodules. The formation of a capsule by a connective tissue membrane is induced by a reactive inflammation in the neighbourhood of the nodule. These nodules are either (*a*) of an embolic origin, and are then situated principally in the periphery of the lungs, the larger ones being at first red, wedge-shaped foci (infarcts), and their structure the same as that of the nodules of glanders on the nasal mucous membrane; or (*b*) they represent lobular-pneumonic foci, in which the alveoli are filled with red and white blood-

* Nocard points out that when glandered horses are treated with mallein, a certain proportion of them recover, in which case the nodules which were present in the lungs cease to contain living glanders bacilli—a fact he has fully proved by inoculation. On *post-mortem* examination of the lungs, the nodules may be readily felt by passing the hand with firm pressure over the surface of the lung, which, when badly diseased, will feel like a bag full of shot or peas.—TR.

corpuscles, and with desquamated epithelium of the lungs. Very soon, disintegration in the centre sets in, and, in the periphery, a reactive inflammation, which manifests itself at first by a dirty-white, lardaceous, shining, inflammatory ring, out of which a connective tissue capsule becomes developed later on. In other cases, the foci suppurate (cavities of glanders); and, finally, glanderous nodules may appear in the neighbourhood of primary foci in consequence of a glanderous inflammation of the lymphatics. Besides these nodules, we find symptoms of chronic bronchitis, peribronchitis, parabronchitis, bronchiectasis, atelectasis, gelatinous infiltration of the tissue of the lungs, and less frequently circumscribed or exudative pleuritis.

Infiltrated glanders of the lungs forms tumours the size of a walnut up to that of a child's head, and consists of a diffuse glanderous infiltration of the alveoli and of the interstitial connective tissue (lobar or greater lobular broncho-pneumonic foci). Frequently the infiltrated parts of the lungs very closely resemble, on section, a soft sarcoma. They are of a dirty-white colour, of a gelatinous juicy consistence, and of an irregular shape. They may either become indurated, so as to form hard, connective-tissue-like new growths (fibroma-like tumours of glanders, according to Gerlach), or they may become gangrenous. In nodular glanders, and in the infiltrated glanders of the lungs, the bronchial glands, and frequently the mediastinal glands, become enlarged, indurated and studded with small foci of glanders.

3. In *glanders of the skin*, the nodules of glanders are found in the papillary layer, in the cutis, and in the subcutaneous and intermuscular tissues. The cutaneous nodules vary in size from a hemp seed up to that of a pea. They suppurate rapidly and form small ulcers. The nodules in the subcutis are inflammatory (metastatic) tumours the size of a pea up to that of a hen's egg. They change into large abscesses and burst outwards. We then find sinuous ulcers with a central cavity (crateriform ulcers), from which a purulent, lymph-like, tenacious secretion discharges. In the neighbourhood of the nodules the lymphatic vessels are inflamed, swollen, and frequently resemble a rosary or knotted cord, as they are divided by nodules of the size of a lentil to that of a hazel-nut. Ulcers often develop from these secondary nodes. The neighbouring lymph glands are at first swollen and soft; but later on they become indurated by the growth of connective tissue and studded with dirty-white nodules about as large as a pin's head, or with

yellow foci of caseation. The capsule around the lymph glands becomes infiltrated with small cells, and subsequently thickened (peri-adenitis and para-adenitis). Only in rare cases do we find secondary chronic farcy, which is marked by a large diffuse new-growth of connective tissue with nodular thickening of the skin. This condition is termed glanderous elephantiasis or pachydermia. It chiefly affects the limbs and head.

4. Among the other organs, the *spleen* most frequently suffers from diseased changes in glanders. It then contains embolic nodules, which vary in size from that of a millet seed to that of a pea, or may be larger, and which usually become calcareous, or may suppurate. Similar nodules occur, though not so often, in the liver, kidneys, testicles, brain, muscles, heart, and bones. In the *bones*, a cellular infiltration of the medulla becomes developed with the formation of nodules, and purulent breaking down of the osseous tissue. Ulcers of glanders are very rare on the mucous membranes of the *eyes*, *stomach*, and *vagina*. The *blood* shows signs of slight leucocytosis, namely, an increase of the white corpuscles. Bacilli are found in the blood only in cases of acute general infection.

The **grey, translucent nodules in the lungs of the horse** have within the last few years become the subject of a lively controversy. In opposition to the customary view, and especially in contradiction to Nocard, Schütz regards the grey, transparent nodules in the lungs of the horse as not glanderous, but simply as of an inflammatory nature, and caused by a parasite, which is also found in the kidneys (entozoïc nodules). In contrast to these entozoïc nodules, the glanderous nodules present small hepatisation nodules (pneumonia fibrinosa miliaris), on the decay of which, *the chromatin of the cell nuclei is still preserved*. This peculiar form of nuclear degeneration, which is referred to by Unna as chromatotexis (nuclear liquefaction), though not regarded by him as pathognomonic of glanders, is, Schütz states, by itself a sure distinctive sign by which the glanderous nature of a nodule in the lung may be satisfactorily differentiated from the entozoïc. In opposition to these views Nocard and Gratia have maintained that, from their researches, the grey translucent nodules are not of exclusively entozoïc nature, but are also present in glanders of the lungs. Nocard found besides the usual entozoïc nodules, miliary ones which are produced by the larvæ of *filaria hæmorrhagica*.

Anatomical Conditions in Acute Glanders.—The anatomical changes in acute glanders consist chiefly in a sero-ulcerous disintegration of the respiratory mucous membrane; in a sero-sanious infiltration of the submucosa, subcutis, and intermuscular tissue; in inflammation and suppuration of the

lymph vessels and lymph glands ; and in metastatic formations in the skin, lungs, etc. The nasal mucous membrane, in particular, is covered with rapidly-spreading ulcers, and shows diphtheritic disintegration, and the submucosa is considerably infiltrated in the neighbourhood of the ulcers. The mucous membrane of the larynx, especially on the epiglottis and ventricles of the larynx, is phlegmonously swollen and covered with ulcers (stenosis of the larynx). Similar changes are found on the mucous membrane of the pharynx. The lungs are studded with purulent metastatic foci or fresh nodules. The skin is excessively swollen and covered with glanderous nodes. Sometimes diffuse gangrene of the skin occurs. Acute glanderous myocarditis has also been observed.

Statistics.—Röll gives the following statistics on the respective participation of the individual organs in glanders ; Among 3,317 glandered horses, 1,529 (about half) manifested the disease only in the nostrils ; 294 only in the lungs ; and 218 only in the skin. In 846 cases, both the nostrils and the lungs were affected at the same time ; in 217 cases, nostrils, lungs and skin ; in 164, nostrils and skin ; and in 49, lungs and skin. In 210 *post-mortem* examinations at the Berlin College, the lungs were found to be free from the changes of glanders only in 10 cases ; and in 173 autopsies at the Vienna College, only in 28 instances. Bollinger found the lungs to be unaffected only on 4 occasions in 52 cases of glanders which he examined *post-mortem*. It appears from this that the lungs in glanders are affected in the large majority of cases. Of the 1,828 horses which were found suffering from glanders in London in 1901, 40 per cent. displayed pulmonary glanders only ; in 38 per cent. the nasal mucous membrane was also attacked ; in 20 per cent. the trachea ; and in 18 per cent. the larynx.

Symptoms of Chronic Glanders.—The early stages of chronic glanders usually escape notice ; for the disease, as a rule, runs a course of months, and sometimes of years. Owing to its insidious approach, entirely wrong views have been taken about its period of incubation, which, according to inoculation experiments, is only from 3 to 5 days.

The first symptom is a unilateral, or more rarely a bilateral *nasal discharge*, which begins in the form of small quantities of dirty white mucus (chronic nasal catarrh). This discharge may fluctuate in amount or may cease for a time. Later on it may consist of a grey or greenish-yellow, discoloured, sticky fluid which comes from the ulcers, and of a clear, limpid or yellowish catarrhal secretion. Sometimes the discharge is temporarily sanious. Such hæmorrhages, which are generally slight, are often the first visible sign of old-standing cases of

glanders. The source of the hæmorrhage is an ulcerous erosion of minute vessels, and the engorgement and rupture of exceedingly small veins. In a few cases, however, the bleeding is more intense, and even a fatal hæmorrhage may occur, especially when one of the larger pulmonary vessels inside a glandered cavity becomes corroded through and is thus opened.

Nodules and *ulcers* may also be found on the nasal mucous membrane, from which they may, however, be entirely absent, or may appear only at a very late stage of the disease. These nodules may also be felt in the inferior part of the nasal septum or in the conchæ, in either of which positions their stay is of very brief duration ; for they rapidly change into superficial or deep-seated ulcers with eroded, banked-up, thickened borders and a lardaceous base (ulcers of the hard chancre type) from which, later on, radiating, star-shaped, or elongated scars originate.

These symptoms are associated with a *swelling of the submaxillary lymph glands*, which is at first somewhat diffuse, more or less hard, doughy, and slightly painful. Later on, it becomes nodular, indurated, painless, and has prominences on its surface. It is usually adherent to the lower jaw, in which case it cannot be displaced. It may also be adherent to the skin. In exceptional cases an abscess, which is generally superficial, forms in the glands. Sometimes the swelling of the glands is absent when the nasal mucous membrane is not infected, and at the beginning of the attack. It is present in almost all cases of glanderous ulceration of the nasal mucous membrane.

The *state of nutrition* becomes visibly impaired, proportionately to the duration of the disease ; the patient loses condition and gets quickly fatigued when worked ; and the coat becomes dry and rough. Frequently, though by no means invariably, the animal has a cough combined with chronic dyspnœa, and appears as if broken-winded (glanders of the lungs).* Not uncommonly the horse suffers from irregular, remittent, or intermittent fever, which is proportionate to the extension of the catarrh and ulcers, and which frequently disappears entirely when the animal is at rest. Sometimes, particularly after exercise, bleeding of the nose is set up by the ulcers eroding the walls of small blood-vessels. Internal

* In England we recognise the fact that grunting, without the horse being a roarer, is a symptom of chronic lung disease, which is important in the diagnosis of glanders only when other symptoms of glanders are present. This remark applies equally well to unthriftiness of coat, fever, running from the nose, etc.—Tr.

bleeding to death and hæmaturia (glanders of the kidneys) has been observed in a few cases. Trasbot remarks on the tendency to fracture (of the ribs) which the bones of glandered horses acquire on account of their becoming brittle. Towards the end, the animal suffers from œdematous swellings of the limbs and lower portions of the abdomen and chest, and also from inflammatory swellings of the joints, testicles, and scrotum.

Glanders of the skin does not occur so often in chronic glanders, as in acute glanders. Its favourite seats are the limbs, shoulders, breast, and hypogastrium. The nodules or boils, which are situated partly in the skin, partly in the subcutaneous connective tissue, vary in size from a pea to a walnut, and may disappear to some extent ("fugitive glanders"); although they generally change into crateriform, sinuous ulcers, the discharge from which is sticky, discoloured, and frequently sanious. Smaller superficial ulcers which are covered with a crust are more rare. The efferent lymph vessels appear to be swollen in the form of a rosary [or knotted cord], the beads of which often become ulcerated [farcy buds]. The affected lymph glands get enlarged and, later on, indurated, or they may form abscesses. It often happens that the animal goes periodically lame from the recurrence of the affection. When, as may occur on rare occasions, glanderous elephantiasis sets in, the skin becomes enormously thickened and the underlying tissues swollen, so that the head, for instance, may assume the appearance of that of a hippopotamus. Besides this, the skin gets studded with nodules, corrugated and extremely hard to the touch.

The progress of chronic glanders is exceedingly slow. The first visible symptom usually appears only at the end of several weeks or even months. It is even possible that glanders may be present for years in an occult state, and that the affection of the nasal mucous membrane or of the cutis may take place from the lungs by metastasis; but the anatomical changes, namely, the nodules in the lungs, may form in a few days. A distinct improvement in the disease after a long rest and with good feeding often takes place. On the other hand, an exacerbation may occur from over-exertion, chill, etc. The duration of chronic glanders may extend to 7 years.

[The use of mallein has proved that a large proportion of glandered horses show no visible signs of the disease. Since the mallein test has been brought into use in England, this percentage has naturally become far

higher than in olden times, when it was the practice to wait for ulcers to form in the nasal passages, before definitely deciding that the animal was glandered.—TR.]

Symptoms of Acute Glanders.—Acute glanders is comparatively rare in horses (about 10 per cent. of all the cases), although it is the usual form among donkeys and their hybrids. It may be the primary form or may develop from chronic glanders which has become generalised or complicated with acute diseases. Inoculated glanders, as a rule, assumes an acute type. Acute glanders is usually a very rapidly progressing septic infective disease, which is accompanied by gangrene of the respiratory mucous membrane and by metastatic formations in the skin, lungs, and other organs.

The affection begins with rigors and high fever, in which the temperature may rise to 108° F., and is associated with a muco-purulent nasal discharge, which, later on, becomes sero-sanious, and which is often mixed with saliva and fodder by regurgitation. The visible nasal mucous membrane is covered with nodules and ulcers which frequently become confluent, so that the entire mucous membrane finally suffers from purulent gangrene and is covered with diphtheritic masses. These changes on the nasal mucous membrane may take place in a very short time (two or three days). The breathing is difficult, sometimes snorting, sometimes rattling, groaning, and " roaring " (stenosis of the larynx). The following symptoms of cutaneous glanders also appear : œdematous swelling of the skin ; formation of nodules and ulcers in the skin ; cord-like inflammation of the lymphatics (especially in the neighbourhood of the head) ; and swelling and suppuration of the lymph glands, particularly those of the intermaxillary space and retropharyngeal glands. There is loss of appetite and frequently difficulty in swallowing, on account of the glanderously inflamed condition of the mucous membrane of the pharynx. Diarrhœa sets in ; the urine contains large quantities of albumen ; and the animal becomes rapidly emaciated and prostrate.

The progress is usually severe, and has an invariably fatal termination in from 3 to 14 days.

Glanders in Cats and Beasts of Prey.—Glanders, when occurring in cats, lions, tigers, etc., after feeding on glanderous horse-flesh, is usually acute. The symptoms consist of a very acute inflammation of the mucous membrane of the nasal cavities, maxillary sinuses, larynx and trachea ; with a discharge of greenish, foul, and, later on, bloodstained, ill-smelling mucus ; great difficulty of breathing (snorting) ; swellings of the lymphatic glands in the trachea ; swelling of the nose ; of the whole

head ; of the legs (lameness) ; formation of nodules and tumours on the skin, with gangrenous mortification of portions of skin as large as the hand ; diarrhœa ; emaciation ; lassitude. Death takes place in from 8 to 14 days after the infection.

Glanders in Man.—The symptoms of glanders in human beings are also of great importance to the veterinary surgeon. Although the disposition of men to the disease is, generally speaking, but small, yet cases of glanders unfortunately are always occurring, especially among veterinary surgeons. Human glanders occurs with special frequency in Russia (Woronzow, Pedkow, and others).

The seats of infection are usually the hands, the nasal mucous membrane, lips and conjunctiva. After an inoculation-stage of from 3 to 5 days, the seat of infection first swells and becomes painful ; then inflammation of the lymphatic vessels and swellings of the lymphatic glands appear. Sometimes, also, the first positive symptoms are those of general febrile disturbance. To these are added, in 50 per cent. of the cases, nasal discharge and ulcers on the nasal mucous membrane, with a very characteristic swelling of the bridge of the nose ; afterwards pustules, abscesses and ulcers of the skin ; ulcers in the cavity of the mouth, in the throat and larynx and on the conjunctiva ; swellings of the joints ; high and continuous fever, with grave general symptoms ; sometimes, also, intense gastro-intestinal derangement. The fatal result generally occurs in from 2 to 4 weeks, occasionally after even a few days ; in other cases glanders becomes chronic, and its duration extends to months and years. In acute glanders, the bacteria are demonstrable in the blood (Wassilieff). Primary glanders of the lung has sometimes been observed in man, as well as in the horse (District Veterinary Surgeon Niebel). The diagnosis is established by—in addition to the symptoms already mentioned—the possibility of inoculation ; on the re-inoculability on horses or guinea-pigs ; and on the demonstration of the bacilli. With regard to the differential diagnosis we have to consider typhus, septicæmia, influenza and articular rheumatism. *Treatment* in cases of generalised glanders is usually ineffective. Only when the affection is a purely local one can we, by deep cauterisation, excision or erosion, succeed in healing the glanderous focus ; as we have had an opportunity of seeing in a case of fresh infection.

According to Galtier, the infection from glanderous horses by the medium of the atmosphere plays but an insignificant part in the case of human beings, so that they run hardly any risk of being infected by inhalation (the case above referred to by Niebel was produced by experimentation with cultures of the bacilli of glanders). The usual mode of infection in chronic glanders is the direct or indirect contact with the nasal discharge and secretions of the ulcers of horses affected with the disease. The infection may also be conveyed by the saliva, the teeth, the bit, and by the dung. In cases of acute glanders all of the organs are virulent, as well as the blood. Of the mucous membranes, the conjunctiva is specially susceptible. The intact skin is not penetrable ; on the other hand, the smallest lesions, scratchings, abrasions, rubbings, are extremely dangerous. With a view to prophylaxis, Galtier recommends the following procedure : 1. The persons who have the charge and attendance of horses affected by, or suspected of, glanders are to be on

their guard against inoculation ; they must not sleep in the stables nor walk on the straw with naked feet, nor lie on the straw ; they must not employ for their own use either the cloths or the other articles used on the affected animals ; they should take care of every lesion and have it directly treated with proper remedies ; and have their hands carefully washed after every manipulation of the animals. 2. In examination, attendance, dressing and harnessing of the animals, whether actually sick or merely suspected, care must be taken of the ejection of virulent matters, and their contact with the face and eyes must be avoided and careful removal by washing carried out whenever it does occur ; injuries of the skin must be directly cleansed by washing ; contact of the eyes or of scratched surfaces with unclean fingers or nails must be avoided ; and after every examination and every operation the hands must be most carefully washed. 3. Before dissection or manipulation of glanderous (or suspected) matter, lesions or scratchings of the skin are to be cauterised with silver nitrate or tincture of iodine ; injuries from instruments or splinters of bone are to be carefully avoided ; when any occur, they must directly receive antiseptic washing, and in requisite cases excision and cauterisation of the injured parts must be carried out ; after a dissection, and often, indeed, during the same, the hands should be washed with an antiseptic solution.

Clinical Diagnosis.—An exact diagnosis of glanders is more important and, under certain circumstances, more difficult than that of any other equine disease. The following aids to the diagnosis of glanders are usually at our command :—

1. Clinical symptoms of the disease.
2. Excision of the laryngeal glands.
3. Artificial production of fever (conversion of chronic glanders into acute).
4. Inoculation in other animals
5. Cultivation of the bacilli of glanders.
6. Mallein.

(1.) Glanders may be very easily diagnosed when the chief *clinical symptoms*, which are as follows, are well marked: a purulent, tenacious and rather clear nasal discharge; nodules and ulcers on the nasal mucous membrane with a lardaceous, dirty-yellow base, and raised, thickened edges ; unilateral, indurated and uneven swelling of the submaxillary glands, which are firmly adherent to the lower jaw ; farcy buds and boils on the skin, with ulcers of the skin and a corded and knotted (rosary-like) condition of the lymphatics ; phlegmonous swellings of the skin without any apparent cause, especially on individual limbs, hypogastrium, under-surface of the chest, scrotum, etc. ; rough and harsh coat ; gradual emaciation ; irregular fever; broken wind ; cough ; periodical bleeding of the nose ; and increasing loss of strength. Frequently, however, only a few of these

symptoms, or even only one is present, so that a correct decision becomes very difficult, and often impossible. In such cases we speak of "suspected glanders"; more especially when a number of suspicious symptoms occur simultaneously. Of the suspicious symptoms, the foremost in importance is a unilateral nasal discharge; though such discharge by itself offers nothing characteristic; frequently a discharge on both sides is found in glanders; while the typically significant greenish-coloured, viscid nasal discharge (intermingled with watery drops) is by no means present in every case of glanders. Suspicious, also, is a hard, longitudinal, painless swelling of the laryngeal lymphatic glands. Such a glandular swelling may, however, be absent in horses suffering from glanders, and may be present in cases of other diseases; *e.g.*, chronic strangles. Even the non-suppuration of the glandular swellings, which was formerly pointed out as a foremost distinctive mark in glanders, occurs in very rare cases (mixed infection). The glandular swellings of glanders are usually associated with glanderous ulcers on the nasal mucous membrane; although the former may occur without the latter. Specially suspicious are gland swellings which disappear spontaneously and then return. The only exact symptom of pathogenic value in diagnosis is the presence of the characteristic ulcers with their lardaceous base, and their eroded, raised edges. It is of great practical importance to search on the mucous membrane under the alæ of the nostrils for these ulcers, which may appear independently of any nasal discharge. We should be careful not to mistake them for accidental wounds, which are often in lines, and covered with a firm scab. The nodules and radiating, star-shaped cicatrices are also of diagnostic value. Scars resulting from injuries are irregular, usually striated and quadrangular. An irregular, remittent or intermittent fever is always suspicious, if it arises without any other apparent cause. Emaciation, unthrifty coat, broken wind, and cough are, undoubtedly, suspicious phenomena, when it is probable that infection has taken place, which fact should be borne in mind by those who own large numbers of horses.

The artificial illumination of the interior of the nostrils with the nasal mirror furnishes us with a valuable optical means for the exact examination of the nasal cavity. It has been used for a considerable time in veterinary medicine; and in many cases furnishes an advantage of the highest importance in forming a decision, as the upper portions of the nasal mucous

membrane could not otherwise be seen. As well as the nasal mirror, the Priestley lamp and the electric light are also to be recommended. An examination of the nasal mucous membrane of substantially more complete character is made with the rhinoscope constructed by Polansky and Schindelka.

Traphining the antrum of Highmore was first proposed in 1851 by Hertwig, and in 1859 by Haubner, as an aid to diagnosis, for which purpose it has been often used since then. It is said that this operation enables us, in cases of the disease, to find glanderous changes in the antrum. Its value, however, is very much restricted by the fact that, in glanders, the mucous membrane of the antrum often remains unaffected. Besides, a non-glanderous inflammation of the mucous membrane of the antrum may produce a thickening effect on it. Also the glanderous changes, mentioned by Haubner, are frequently absent from the wound caused by the operation, even when glanders is present in the immediate neighbourhood.

(2.) **The extirpation of the tracheal lymphatic glands** (Haubner and Bollinger), with the object of examining them anatomically, is by itself of doubtful use, for the following reasons: The characteristic foci of glanders may be absent from them; simple induration of these glands also occurs in chronic nasal catarrh; in attacks of strangles, purulent foci may be disseminated in the glandular tissue; and sarcomatous and carcinomatous metastatic deposits may be found in these glands. On the other hand, it is very easy to examine bacteriologically the glanderous tubercles found in the parenchyma of the gland, so as to demonstrate the presence of the bacilli. We may consequently conclude that extirpation of these glands, combined with their bacteriological examination (cultivations on potatoes), is a valuable diagnostic means with living animals (Rieck, Chelchowski, and others). According to Rudenko, the bacilli of glanders can be found in the lymph glands, even when these glands are but little, if at all, enlarged.

Puncture of the lung with a Pravaz syringe introduced through an intercostal space, for the purpose of aspiration of some of the glanderous pulmonary fluid and bacteriological examination of the same, has recently been adopted by Catterina. Noniewitsch recommends a withdrawal of blood, after a preliminary mallein infection, with subsequent bacteriological investigation.

(3.) As far back as 1843, Bouley proposed the *artificial production of fever* as a means for hastening the manifestation of symptoms in cases of latent glanders. This procedure is

founded on the long-established fact that the presence of fever tends to render the course of chronic glanders more or less acute. In recent times, Lustig recommended for this object that horses suspected of glanders should be made to work hard, and that attention should be paid to variations in their temperature, and also to the occurrence of bleeding from the nostrils. Cagny and others state that subcutaneous injections of oil of turpentine have the effect of changing chronic glanders into acute glanders. The diagnostic intravenous injection of *argentum colloidale* (0.5 : 50), recommended by Dieckerhoff (with the view of converting chronic glanders into acute), has not proved satisfactory (Marek, Arndt, Blome, Peters, and others).

Auto-inoculation (malleosation).—The inoculation of suspected animals on the abraded nasal mucous membrane with their own secretions, such as the discharge from the nostrils or from ulcers—was formerly often used to verify diagnosis. The results, however, varied greatly. In very many cases treated by Bagge, Tscherning, St. Cyr, and others, the experiments resulted in a merely local and narrowly-restricted glanderous process, or the inoculation failed entirely. It is evident that glanders causes more or less, though by no means absolute, immunity to auto-inoculation.

(4.) **The inoculation of other animals** is certainly the most valuable diagnostic means at our disposal. For this purpose, asses, horses, and guinea-pigs are the most suitable. Dogs are less so; and rabbits least of all. Löffler considers the field-mouse to be the best subject for inoculation experiments with pure cultures. Kitt gives the preference to the vole [*arvicola terrestris*] and hedgehog. The ass, if it were less expensive, would be the best of all for practical purposes; because in him glanderous inoculation is almost always followed by the acute form of the disease, which then runs a fatal course in 8 or 10 days. Next to the ass the horse is the most suitable. We must remember that negative results must not be accepted unconditionally; because the material used for inoculation may have been free from bacilli, and because the experimental subject, especially if it be an old horse, may have been suffering from latent glanders, and, on that account, may have failed to react. Horses, as a rule, are too costly for this purpose.

We recommend *guinea-pigs* on account of their cheapness and certainty of the inoculation taking effect. These animals, after virulent inoculation, manifest all the characteristic symptoms of glanders; one of the most typical in males being

inflammation of the testicles. Male guinea-pigs are consequently particularly good subjects for these experiments. Their employment, however, possesses the drawback that, in their case, the average duration of the experiment will be as long as a month. Löffler recommends the following method for inoculation. The hair is removed from the lateral surface of the abdomen, and the skin is pinched up into a fold and cut with a pair of [rowelling] scissors through its entire thickness, so as to make a gaping wound of about 0·4 inch in length. A previously heated steel needle is inserted into the wound, and is moved to and fro under the skin so as to form a pocket, which serves as a receptacle for the inoculation material. For this purpose we prefer to use the discharge from the glanderous ulcers, either in the form of pure pus or lumpy masses. It is well to inoculate three or four guinea-pigs for each experiment. In such cases, the first symptom is redness of the edges of the wound, and the formation of a round or oval ulcer with a suppurating, infiltrated base and a thickened edge. Towards the end of the first week the neighbouring lymph glands swell to the size of a pea, and even up to that of a chestnut, and form abscesses. Sometimes the entire process heals spontaneously from this stage. More frequently, however, inflammation of the testicles takes place during the second week, with the formation, in the testicles, of nodular spots, which develop into abscesses and discharge pus rich in bacilli. These symptoms may be associated with purulent arthritis, and cutaneous or subcutaneous nodules, which suppurate or burst. When they occur on the face, they frequently take their origin from the periosteum of the bones, and sometimes eat through, into the nasal cavity. Finally, the nasal mucous membrane, becomes affected; the animal sneezes, and breathes with difficulty; a discharge, which dries up into crusts, issues from the nostrils; and death takes place generally in the third or fourth week under circumstances of great emaciation and exhaustion. Chronic glanders may also develop with caseous foci in the lungs, heart, liver, spleen, lymph glands, etc., and then runs a course of from 2 to 4 months. Chronic indurated glanders in guinea-pigs may sometimes heal spontaneously. Besides the described changes, we find, *post-mortem*, nodules in the lungs, spleen, omentum, testicles, and retro-peritoneal tissue; ulcers in the nasal mucous membrane; perforation of the septum nasi; and partial destruction of the facial bones. The blood contains no bacilli, contrary to what is the case in the organs affected by glanders. (By the intra-

peritoneal inoculation which has recently been recommended, a glanderous testicular inflammation is often produced after 8 days; these inoculation-methods have, however, a mixed infection for result in many cases.)

The intraperitoneal inoculation of guinea-pigs, which has been recommended by Strauss and others, produces, indeed, the above-described changes more rapidly; inasmuch as even after from 2 to 3 days a typical affection of the scrotum appears; but it frequently causes a mixed infection and fatal septicæmia, for which reason the subcutaneous method is to be preferred (Kitt, Tröster).

Inoculation experiments with negative results are not demonstrative of the absence of glanders, as the inoculated secretion may, in some cases, be free from bacilli. According to recent researches, guinea-pigs sometimes, after inoculation, display the symptoms of pseudo-glanders (see below), and of glanders-like strangles; and, accordingly, it is recommended in cases of positive results, to confirm the diagnosis by the production of potato-cultures from the pus of the animals examined.

Subjects for Inoculation.—The **dog** has also been recommended as a suitable subject for inoculation (Pütz, Galtier, Reul, Molkentin, and others). A seton saturated with infectious secretion is placed under the skin of the nape of the neck. On the third day an ulcer, resembling a hard chancre, with a purulent base, hard edge, and irregular shape, appears on the site of the inoculation and gradually becomes enlarged with marked symptoms of constitutional disturbance. We cannot form a positive diagnosis from these symptoms, which, in the majority of instances, are the only ones present. In rare cases, farcy buds and ulcers appear on other parts of the skin; the joints, especially those of the hind limbs, become attacked by acute inflammation, and the patient suffers from hæmorrhagic diarrhœa, purulent conjunctivitis, ulcerous keratitis, emaciation, etc. According to a personal communication by Neimann, inoculation experiments in 300 cases gave only 4 negative results. Dogs are not very suitable subjects for inoculation; because, when infected by glanders, they rarely manifest general symptoms, and in their case it is always difficult to form a correct judgment from the condition of the cutaneous ulcers.

Lisitzin and Buchner prefer the **cat** to the dog as a subject for inoculation. These animals are inoculated on the upper surface of the neck, and show from the third day a swelling at the site of the inoculation. After 5 to 7 days a typical glanderous ulcer appears in the neighbouring tissue by metastasis. The cats which died in 2 or 3 weeks showed, *post-mortem*, glanders of the nose and lungs. Peuch transmitted glanders to sheep from donkeys.

Rabbits, though formerly much used, **are unsuitable**; because they

die, after being inoculated, more frequently from septicæmia than glanders. Besides, as Friedberger has proved, a period of 2 or 3 months is required for observing them : also, the results they give are not uniform. If, after saturating the inoculation threads with nasal discharge, we draw them through the skin of the cartilage of the inside of the ear, there will form at first, purulent boils the size of a hemp seed up to that of a pea, and irregular ulcers with an uneven and suppurating base and raised edges. These ulcers sometimes perforate the cartilage of the ears. They may, however, heal spontaneously. Friedberger considers that these changes are not very characteristic. Later on, if the inoculation be successful, a discharge will issue from the nostrils, and death take place after about 2 months. On *post-mortem* examination the lungs and spleen are found to be stuffed with glanderous nodules.

Löffler considers **field-mice** to be very good subjects for inoculation with pure cultures of the bacilli of glanders. They, however, die of septicæmia when inoculated with the nasal discharge. Death takes place in 3 or 4 days after the inoculation with pure cultures. On the first, and sometimes even on the second day, they remain apparently healthy. After that, they lose their vivacity, their hair stands on end, they cower together, refuse to eat, breathe heavily, and suddenly fall down dead without any preceding convulsions. On *post-mortem*, we find on the site of the inoculation a greenish or dirty-white infiltration, from which the swollen and corded lymphatic lymph vessels proceed to the enlarged lymph glands that are infiltrated with numerous pale yellow small foci. The liver is stuffed with numbers of grey nodules, which are absent from the lungs, and which are very rich in bacilli. The nasal mucous membrane and skin are in a normal condition. The inoculation, as in guinea-pigs, is made into a small pocket on the back. Besides the field-mouse, Kitt finds the **vole** (*arvicola terrestris*) suitable for pure cultures. In this animal the spleen and inguinal glands are most frequently affected. Kitt also recommends for this purpose the **hedgehog** (*erinaceus europaeus*), which becomes affected with very characteristic glanders of the spleen and lungs. Kranzfeld and Grünwald inoculated the **earless marmot** (*spermophilus guttatus*) with glanders.

(5.) The **bacteriological proof of the presence of the bacilli** is the most certain diagnostic means ; but it can seldom be carried out in practice. Only on unopened glanderous foci (glanders of the skin, pustules of the nasal mucous membrane at the entrance of the nostrils, and in the lymph glands) can we succeed, under all necessary precautions, in obtaining a characteristic culture of bacilli on serum (minute, yellow, translucent drops), or on potatoes (a honey-coloured coating). Of all these means, the only practical one is extirpation of the submaxillary glands. Cultivations can be adopted for diagnostic purposes only in exceptional cases, as for instance in veterinary colleges (Kitt). An examination of the nasal discharge or of the secretion of an ulcer for bacilli is, as a rule, without result ; because the bacilli of glanders which are in them do not possess

any characteristic staining. The above-mentioned secretions swarm with numerous other bacilli which stain in exactly the same manner as the bacilli of glanders. As these secretions are also more or less decomposed, it would be very troublesome to prepare a pure cultivation from them. Consequently, the inoculation of animals, is, as a rule, the only practical diagnostic means which remains to us.

Methods of Staining the Bacilli of Glanders.—The technique of staining the bacilli of glanders is so unreliable, troublesome, and tedious that the practical veterinary surgeon will hardly ever undertake it (Kitt). The following methods are recommended. 1. Löffler's first way of staining: the cover glass preparations are made in the usual manner (the material rubbed down on the glass, dried in the air, and passed 3 times through a spirit flame); placed from 5 to 10 minutes in a solution of methylene blue (8½ fluid drs. concentrated alcoholic solution of methylene blue mixed with 3½ oz. of a $\frac{1}{100}$th per cent. solution of caustic potash); held for a few seconds in a 1 per cent. solution of lactic acid; rinsed in water; and examined. To make the cover glass preparation, the cover glass on which the secretion or other material is spread out is dried by being drawn slowly through a spirit flame, and is then placed for 5 minutes in the staining fluid, which consists of equal parts of aniline water, solution of gentian violet, and solution of caustic potash (1 to 10,000), after which it is dipped for one second into a 1 per cent. solution of acetic acid which has been stained a hock-yellow by the addition of a solution of tropaolin, and is immediately afterwards rinsed in distilled water. 2. Löffler has recently begun to stain the bacilli of glanders as follows: Koch-Erlich's gentian violet-aniline water solution is mixed with an equal quantity of a solution of caustic potash (1 to 10,000), or of a 0·5 per cent. solution of ammonia. The cover glass or the preparation is placed for about 3 minutes in this mixture, and is then put for not longer than 1 second in a 1 per cent. solution of acetic acid, to which as much of a watery solution of tropaolin has been added as will make the fluid assume a light straw colour, and the cover glass or the preparation is then immediately rinsed in water. 3. According to Sahlis, the cover glass, after having been duly prepared, is placed for 5 to 10 minutes in a mixture of a 1 per cent. solution of methylene blue and of a 1 per cent. solution of borax, after which it is rinsed in water or in dilute alcohol and dried. 4. Other methods are those of Kühne and Noniewicz. 5. In contrast to other similar bacilli, those of glanders do not stain by the Gram method.

(6.) **Mallein,** which is a preparation made from the bacilli of glanders, was first manufactured and investigated in 1891 by Kalning and Hellmann, as a means for diagnosing glanders, analogous to Koch's tuberculin. The numerous experiments which have been made with mallein during late years in Germany, Austria, France, England, Russia, Denmark, Belgium, and other countries, have not yet resulted in a conclusive decision

respecting its value. The reported results of the diagnostic value of mallein in glanders are very contradictory. This contradiction may possibly be explained in part by the use of preparations of mallein of unequal value. Especially does the fluid mallein of Preusze appear to be an unreliable preparation, and of little value, as compared with the dry mallein.

1. According to Preusze, Heyne, Schilling, Peters, Felisch, Dieckerhoff, Lothes, Gutzeit, Foth, Schindelka, Johne, Kitt, Höflich, Feist, Hutyra, Preisz, Nocard, Laguerrière, and others, mallein is for glanders a diagnostic agent similar to tuberculin for tuberculosis. If a horse receives a subcutaneous injection of 0.05 (= 0.77 grains) of malleinum siccum, corresponding to 0.5 (7.7 mins.) of crude mallein (the injection being administered in the evening, the fall of temperature takes place on the following morning), and displays a rise of temperature of over 3.6° F.—with typical double fever curve (Schindelka), the animal is decidedly glanderous, and should accordingly be slaughtered forthwith. On the other hand, a horse is surely free from glanders when the rise of temperature ranges below 2° F. For rises of temperature ranging between 2°–3½° F. the case remains doubtful; and a second (or even third) injection may be administered after from 2 to 3 weeks. It must here be prefaced that the body temperature at the time of the injection must be between 99° and 101° F.; feverish horses are not suited to mallein inoculation, and in temperatures of 99°–99.5° F., a higher reaction must develop (4.5°–5.4° F.) before diagnostic certainty can be attained. The local swelling and the general disturbance which occur after the injection in horses are of less significance, or, indeed, of none at all (Foth, Johne, Schindelka).

2. According to Schütz, Olt, Leblanc, Mehrdorf, König, Tröster, Joest, Pötschke, Krajewski-Borella, Wirtz, Bianchi, Bruschettini, Robeis, Schöneck, Peters, and others, mallein possesses no specific influence in glanders, and is, accordingly, unreliable as a diagnostic agent. In this respect the following may be said:

(*a*.) Many horses, healthy and free from glanders, have reacted typically to mallein, and with high temperatures. Of 52 suspected horses, 15 displayed a rise of temperature of 2⅘° F., and over; 7, of 3° F.; 30, of 0–1.8° F. None of these horses, when slaughtered, were found to be affected with glanders (Schütz). It is not improbable that, in many cases, horses which proved to be glanderous on dissection, displayed

non-glanderous nodules in the lungs (Schütz, Olt). Similar observations of rises of temperature in absolutely healthy horses have been made in the Prussian cavalry by Mehrdorf, Deppe, Walther, Peters, Engelen, Scharmer, Tiede, and others.

(*b.*) Horses actually affected with glanders do not always react to mallein (Siedamgrotzky, Olt). In this connection, the inoculations carried out by Olt in Mgowo are very instructive. There, 9 non-glanderous horses reacted typically, while 3 which were suffering from the disease did not react. Thereupon Olt concludes that, consequently, the mallein of Preusze is not a means of proving the disease of glanders among horses. The argument that after the injection glanders is sometimes cured cannot be regarded as conclusive.

(*c.*) Horses affected with other diseases sometimes react in much the same way to mallein as do those suffering from glanders. Of the diseases in which mallein develops a typical rise of temperature the following have been established: emphysema, pneumonia, bronchitis, lymphadenitis, dental caries, alveolar periostitis, malanosis, actinomycosis, botryomycosis, sarcoma, articular inflammations, painful surgical affections.

Mehrdorf, Hendricks and others have drawn attention to the danger of infection of healthy horses with glanders from bad preparations of mallein.

Serum Diagnosis.—In the same way as the serum of typhoid human patients exercises an agglutinating influence on typhus bacilli (Widal's reaction), does the blood-serum of glanderous horses produce agglutination of glanders bacilli (Méry, Wladimiroff, McFadyean, Jensen, Rabieux, Schütz, and others). The agglutination test is usually carried out by placing a measured quantity of cultures of the glanders bacilli in a test-tube, mixed with the blood of the horse suspected of glanders which is to be tested, and allowing the tube to remain undisturbed for some hours. If the blood contains agglutinin, the glanders bacilli sink in form of a viscid, flaky sediment; while in other cases, the fluid remains clear. Accordingly, the test can be carried out only in the laboratory, as a supply of cultures of the bacilli of glanders and the serum of a healthy horse are necessary (Kitt). There is also a deficiency of experiments on the question as to whether the blood of non-glanderous horses, or horses affected with other diseases, does not also agglutinate. According to Jensen, the agglutination test appears to give a very much more reliable result than does the mallein test, and forms a valuable complement of the latter. He thinks that the number of experiments is, never theless, too small to form a definite decision. According to Rabieux the serum of healthy horses does not produce agglutination of the glanders bacilli. The diagnosis of glanders is assured in every case in which the serum agglutinates the glanders bacilli, even after a dilution of 1 : 1,000. The agglutination test forms, in doubtful cases, a valuable completion

of the other diagnostic methods; it will, however, not obtain a general introduction into practice, but must always remain a laboratory experiment.

Clinical Differential Diagnosis.—Glanders may be mistaken for a great number of diseases, of which the following are the most important :—

1. *Simple chronic nasal catarrh* gives the most frequent cause to suspect glanders; because its course is characterised by chronic nasal discharge, swelling of the glands, and sometimes with superficial ulcers. On that account, it was formerly regarded as the first stage of glanders. A considerable time for observation is always necessary to distinguish positively between the two. Simple chronic catarrh is, as a rule, accompanied by no scars on the mucous membrane, and can be permanently cured by local treatment. Inoculation in other animals will conclusively decide the point. Cultures on potatoes have only a doubtful value.

2. The *chronic catarrh of the accessory cavities*, especially of the maxillary sinuses, and of the guttural pouches, is frequently mistaken for glanders. Here also a long time of observation is often necessary. Besides, the curability of the affection by trephining, etc., must be taken into consideration. In cases of caries of the teeth, an examination of the teeth and of the odour (whether healthy or fœtid) from the mouth will decide the point. A case observed by Siedamgrotzky shows that caries of the teeth with chronic inflammation of the maxillary sinuses and glanders in these sinuses may be present at the same time. The presence of necrotic and tumour-like processes of the bones in the neighbourhood of the nasal cavity (ethmoid bone, etc.) may lead to a mistake. Inoculation is specially important here.

3. In *strangles* we frequently find the development of lymphangitis with formation of ulcers on the head, throat, etc. The rapid healing of the lesions and other symptoms of strangles generally render the diagnosis easy. The differentiation between glanders and chronic metastatic strangles is more difficult. Here, inoculations and pure cultivations will decide the matter. Rabe has described a case of ulcerous metastatic strangles which was very hard to diagnose, and showed ulcers in the nostrils, palate, pharynx, and guttural pouches. Inoculations in guinea-pigs were followed by death. When the inoculated animals were bacteriologically examined, there were found in them rod-like bacteria which could not be morpho-

logically distinguished from the bacilli of glanders. The micro-organisms were proved not to be the bacilli of glanders, only by cultivations on potatoes and by inoculation in white mice. Rabe consequently considers it absolutely necessary, after having obtained a positive result with inoculation in guinea-pigs, to make cultivations and to inoculate them in white mice in order to be certain of the diagnosis.

4. *Follicular ulceration* of the nasal mucous membrane heals rapidly and without cicatrisation. It spreads to the skin in the neighbourhood of the nostrils, and is usually complicated only with a slight swelling of the glands.

5. *Stomatitis contagiosa pustulosa* is localised chiefly on the mucous membrane of the mouth, and is distinguished by the fact of its course being benign.

6. *Injuries of the nasal mucous membrane* from external influences such as hot steam, smoke, foreign bodies, finger nails, etc., can easily be recognised by an exact examination. The subsequent cicatrices are distinguished by being generally situated on the lower end of the nasal cavity (*septum nasi*). They are oblong, striated or angular, and occur frequently in the same position on both sides. They are flat, hollowed-out, and sometimes surprisingly thick and prominent. Traumatic cicatrices have very rarely a radiating appearance.

7. *Simple bleeding of the nose*, in consequence of traumatic causes, aneurisms, angiomata, phlebitis, varicose veins, etc., of the nasal mucous membrane; hyperæmia of the head; hyperæmia of the lungs; and inflammation of the lungs, may be distinguished by an exact examination, which may have to be continued for some time. Compare also with hæmorrhage of the lungs.

8. *Carcinomata, sarcomata, actinomycosis, melanosis*, and other *new growths* of the glands of the intermaxillary space and other lymph glands. In these cases, extirpation and microscopical examination of the tumour taken in conjunction with the negative result of the potato cultivation will decide the question.

9. *Leucæmia* has sometimes a great similarity to glanders in its clinical aspect: emaciation, debility, swelling of the glands, bleeding of the nose, intermittent fever, swelling of the extremities, and of the epigastrium, etc. The differentiation is frequently possible only by an exact examination of the blood, in which a surprisingly large increase (characteristic of leucæmia) of the white corpuscles will be found. In glanders, on the contrary, the increase in the number of leucocytes is but moderate;

Inoculation is valueless; for in leucæmia no morbid secretion is available.

10. *New-growths in the nasal mucous membrane* can, in many cases, be proved only by *post-mortem*. Among these we may include angiomata of the septum nasi; sarcomata and carcinomata of the nostrils and accessory cavities; enchondromata; rhinoscleromata; osteomelanomata; polypi; and benign chronic fibrous-hypertrophies of the mucous membrane—the last-named often closely resemble infiltrated glanders.

11. *Parasites* in the nasal cavity (*pentastoma tænioides*, and *gastrophilus nasalis*) sometimes produce the aspect of glanders in the nostrils.

12. *Lymphangitis* (simple inflammation of the lymph glands) is frequently mistaken for farcy and cannot always be easily distinguished from it. The fact that lymphangitis develops usually in the neighbourhood of wounds does not always make the diagnosis certain; because glanders may also develop from wounds, and because the primary injuries in cases of lymphangitis may have healed up and disappeared. It is specially important to remember that lymphangitis is usually a local affection; that farcy is a symptom of general glanders; that lymphangitis frequently progresses very acutely with fever and great tendency to the formation of abscesses in the lymph glands; and that farcy usually progresses slowly, without fever and with but slight participation of the glands. The abscesses which appear in the progress of lymphangitis have smooth edges and heal rapidly by granulation; but the abscesses of glanders are crateriform non-healing ulcers. In farcy, the cords of the affected lymph vessels are narrower, as a rule, than in lymphangitis, in cases of which the neighbouring tissues generally participate in the gelatinous infiltration. To make sure of the diagnosis, an inoculation or the cultivation of the bacilli is often indispensable.

13. *Phlegmonous inflammations of the skin* on the extremities (elephantiasis), head and other parts of the body, and pyæmia, especially when it is metastatic, may give rise to a suspicion of glanders. Here also we have to depend on inoculation, because the discovery of the starting point (wounds, bruises, etc.) of the inflammation is not enough.

14. *Urticaria* is fugitive, and causes neither the formation of abscesses nor the swelling of glands. The tumours of the skin which it gives rise to are benign. The same remark applies to *eczema nodosum*.

15. *Petechial fever* is distinguished from acute glanders chiefly by the slight amount of fever that is present, and by the fact that the extensive swellings of the skin are never nodular.

Anatomical Differential Diagnosis.—As pathological anatomy cannot be discussed in a text book of special pathology, we shall content ourselves by briefly mentioning the principal pulmonary changes for which glanders of the lungs may be most readily mistaken. We may, however, state that the macroscopical or microscopical result does not always suffice for a correct diagnosis; but that, very frequently, the clinical and etiological conditions have also to be studied, and the bacilli sought for.

1. The lungs of old horses frequently contain, in greater or less number, calcareous or caseous encapsuled nodules the size of a lentil up to that of a pea, which contain either embolic lifeless larvæ of parasites (*echinococcus*, *sclerostomum*, etc.), and which are to be looked upon as a *phlebitis obliterans nodosa* (Olt) or as pneumonia due to very minute foreign bodies or to inhaled dust (*chalicosis s. nodosis pulmonum*). They may be distinguished from the nodules of glanders—for which they are often mistaken—by the fact that they are all about the same age; by the absence of other glanderous lesions (especially by the absence of the enlargement of the bronchial glands), and possibly by the demonstration of particles of plants, etc., in the nodules. Csokor, Kitt, and others state that true nodules of glanders do not become calcareous. Confirmation of this statement would greatly facilitate the anatomical differential diagnosis of glanders.

2. The embolic and, as a rule, subpleural nodules (varying in size from a pea to a hen's egg) which occur in the lungs of animals suffering from strangles, pyæmia, etc., and which are sometimes scattered over the entire lungs, are mostly of the same size and age, and appear at first as wedge-shaped hæmorrhagic infarcts. As the embolic nodules of glanders possess the same characteristics; proof of the absence of other glanderous changes or the demonstration of a non-glandered primary centre will be required to decide the point.

3. Tuberculosis of the lungs is distinguished first of all by the difference between the respective bacilli. Those of glanders, according to Csokor, are broader than tubercle bacilli and seem to be composed of small and alternately light and dark cubes. Csokor further states that the nodules of glanders consist almost

method, and furnish other cultures. Kutscher has discovered a similar pseudo-bacillus in horses, which produces testicular inflammation in guinea-pigs. In a case of pseudo-glanders of the lung occurring in a horse, Frothingham found a blastomycotic new-growth produced by the yeast torula.

[As glanders is largely imported into the United Kingdom from abroad, no horse should be allowed to be landed in this country without being examined by a veterinary surgeon, and certified to be free from this disease, except in the case of an animal brought from a glanders-free country, like Australia, and in that of race-horses, which very rarely suffer from this disease. Mr. H. G. Rogers wisely suggests the advisability, in this connection, of applying the mallein test, with a quarantine of 3 or 4 days.

With respect to the *prophylaxis* of glanders, the French method (*Journal of Comparative Pathology*, Dec. 1897, pp. 295, 296, and 345), which is as follows, is well worthy of adoption in this country, always taking into account that, under certain conditions, mallein is not infallibly diagnostic.

1. All horses presenting symptoms of apparent glanders should be tested with mallein, and if they show a reactionary rise of temperature and swelling at the site of injection, they should be destroyed. If they fail to react, they should be considered free from the disease.

2. All horses which have cohabited with glandered horses should be tested with mallein, and should be divided into two classes, namely :— (*a*) Those which give no reaction, and which consequently should be regarded as healthy as far as glanders is concerned. (*b*) Those which react, and which, on that account, should be rigidly segregated, and should be again tested with mallein once every month, or every 2 months. If any of this second division, after reaction, develop visible signs of glanders (including those of farcy), they should be promptly destroyed ; and all the others which have ceased to react for 2 successive injections should be declared free from this disease.

Nocard justly points out that the manifestation of external signs of glanders is the last stage of the disease, which then has become general, and has thoroughly overcome the power of resistance of the system. Hence the treatment of such cases, even by mallein, is generally hopeless.

At present, mallein is largely employed in England for the unintentional dissemination of glanders, by owners who use it to find out the condition of their horses, and who, on obtaining the required information, sell those that react. It is almost needless to say that infected animals which manifest no outward signs of the disease, are the most potent of all means for spreading it.—TR.]

BOVINE PLEURO-PNEUMONIA, ETC.

Bovine pleuro-pneumonia — Septic pleuro-pneumonia in calves — Infectious pleuro-pneumonia in goats — Epizoötic inflammation of the lungs in American oxen.

History.—The first account of the appearance of pleuro-pneumonia dates from the end of the seventeenth century. It is stated that pleuro-pneumonia was observed in Hesse for the first time in 1693, and that its earliest appearance as an epizoötic was at the beginning of the eighteenth century, when it prevailed chiefly in Switzerland and the neighbouring countries of Würtemberg, Baden, and Alsace. We possess detailed reports on it from the year 1743. In 1751 the Sanitary College of Zürich published for owners of cattle a guide containing the chief characteristics of the disease. In 1773 the celebrated natural philosopher Haller published an essay on this disease, which was raging severely at that time, and described the symptoms, anatomical conditions, and protective measures laid down by the sanitary police. In 1735 the disease appeared in England; and in 1765 in France. From 1790 it spread over the whole of Germany, France, and Italy; and at the commencement of the nineteenth century, over all the countries of Western Europe. Between 1840 and 1850 it spread to America, Africa (Cape Colony), and Australia.

Etiology.—Pleuro-pneumonia is an infective disease peculiar to cattle. It cannot be transmitted to any other species of animal, except under certain conditions. It may be described generally as an infective inflammation of the lungs and pleuræ (*pleuro-pneumonia boum contagiosa*). The infecting medium, according to Nocard and Roux, is a micro-organism of extraordinary minuteness, much smaller than the smallest known bacteria, the form of which cannot be sufficiently recognised in even stained preparations (punctiform microbes), and which cannot be cultivated on any of the nutritive media hitherto employed. To separate it from all other bacteria, the lymph is passed through a Berkefeld's filter or a Chamberland's bougie, which allow only the microbes of contagious pleuro-pneumonia to pass. The latter are easily cultivated in collodion sacs which are introduced into the abdominal cavity of the rabbit. Its cultivation can also be carried out in Martin's peptonised broth,

which contains 1 : 20 of rabbit or cattle serum ; where it forms excessively small, translucent colonies (opalescence). It may also be grown on solid nutritive media (agar with some drops of serum on the surface).

The infection may be introduced into a cowshed either by diseased cattle, or, less commonly, by bearers such as cattle-dealers, attendants, utensils, fodder, dogs, etc. The sheds in busy cattle markets are very dangerous centres for the dissemination of the disease, which, for this reason, is enzoötic in sugar-beetroot districts, where, on account of the great abundance of wash and beetroot parings, there is a large demand for, and traffic in, cattle. All cattle are not equally susceptible to the contagium. It is generally supposed that about 1 in 4 is immune. The disease is spread in sheds and farms principally by the respired air. Infected cattle can transmit it even during the period of incubation, before any symptoms are apparent. The disease is particularly infectious when it is at its height. The animal remains capable of transmitting the disease for 8 or 10 weeks, or even longer, after the infection, especially when necrotic foci remain in the lungs ; similar to what happens in contagious pleuro-pneumonia of the horse. Walley estimates the duration of infectiousness in cases of encapsuled necrotic foci to be as long as 15 months. The disease may be conveyed by the respiratory air for as great a distance as 40 yards or more. As already stated, it is often carried by attendants, fodder, dogs, etc. ; and more rarely by the flesh of slaughtered animals. This flesh, as a rule, seems to lose its infectiousness on becoming cold. In exceptional cases, the contagium is transmitted from the pregnant cow to the fœtus.

The contagium possesses considerable resistance. It may continue virulent in infected sheds for several months, and even over a year. Laguerrière proved by inoculation that portions of the lungs of cattle suffering from pleuro-pneumonia retained their infectiousness for a whole year at a temperature between 41° and 43° F. The serous fluid of the diseased lungs, when taken in a perfectly pure condition and preserved in sealed glass-tubes, loses its virulence only after two months.

The *period of incubation* is on an average from 3 to 6 weeks ; the minimum being 8 days, and the maximum 3 months. Animals which have once passed through the disease remain immune either for several years, or for the remainder of their lives.

* **Bacteriology.**—Nocard and Roux, in 1898, after fruitless researches lasting a whole year, succeeded in demonstrating bacteria of the minutest size as the causative agent in pleuro-pneumonia; and in producing the typical form of the disease by inoculation with the same. As the culture did not succeed on the ordinary media, and the specific bacteria were also not demonstrable by the usual staining methods, as a last resource, the plan was adopted of placing the cultures, by the aid of collodion capsules, within the bodies of living animals. Very thin collodion capsules, filled with broth and a trace of virulent lung fluid, were placed in the abdominal cavities of rabbits. Through the walls of those capsules bacteria could not pass outwards, nor white blood corpuscles inwards (phagocytosis). On the other hand, the micro-organisms could multiply within the caspules without hindrance; while they absorbed the nutritive fluids, suitable to them, from the body of the inoculated animal. On the other hand, their toxins could diffuse outwards through the wall of the capsule, and produce the characteristic phenomena of poisoning in the experimental animals. The pleuro-pneumonic collodion capsules placed in the abdominal cavities of rabbits contained, after an interval of from 15 to 20 days, a somewhat turbid, milk-white fluid, in which a magnifying power of 2,000 detected innumerable refracting, mobile points of the minutest size. All experiments made for the purpose of producing the natural disease by means of the microbes which were found, have failed (feeding, inhalation, injection—subcutaneous, intravenous, intramuscular, intrapulmonary, intraocular, intracerebral, and syringing into the udder). Only in 4 cattle have Nocard and Roux produced the appearances of contagious pleuro-pneumonia, by the inhalation of pure cultures. The inoculation with cultures on the tail produced the same phenomena as the lymph from the lung, and likewise established immunity. By syringing into the udder, mastitis was produced; with permanent hardening and agalactia (suppression of milk). Instead of milk a thick, pus-like fluid was secreted, which contained the pleuro-pneumonic factors nearly in pure culture, and in a high degree of virulence.

The micrococci formerly found by Pöls and Nolen, and the bacilli liquefaciens which was indicated by Arloing as the productive agent in pleuro-pneumonia, are, accordingly, not to be regarded as the causes of this disease.

Occurrence.—Next to rinderpest, pleuro-pneumonia is the most dangerous bovine disease. It may occur, especially at the commencement of an outbreak, sporadically; although it usually manifests itself enzoötically, and not rarely epizoötically Its presence in certain districts is constant, as in Austria, France, England,* Belgium, and in some parts of Germany, America, Africa, Asia Minor, and Australia. The Eastern European countries, as for instance, Russia, are comparatively free, with the exception of the Western frontiers and sea-ports. The disease is most frequently met with in large herds in which there

* No case of bovine pleuro-pneumonia has occurred in Great Britain since 1898.

is a constant change of cattle, and along great thoroughfares. Countries which do not import any cattle are usually free from the disease.

In Germany, according to the geographical representation of the distribution of pleuro-pneumonia in the official publications on State sanitation, the principal focus of this disease is in mid-Germany; and specially in the rich lowlands lying north and east of the Harz Mountains, between the Saale or Elbe, and the Leine, which are cultivated with sugar-beet and are richly supplied with sugar-factories and the associated industries. The circuits of Magdeburg and Merseburg; also Hildesheim, Lüneburg, Brunswick and Anhalt are especially plagued. The worst affected individual districts of the mid-German foci are Wolmirstedt, Helmstedt, Neuhaldensleben, Aschersleben, Wanzleben, Halberstadt, Mansfelder, Seekreis, Kalbe, Zeitz. Of all the cases occurring in Germany, from 75 to 90 per cent. come from these mid-German plague foci.

Pleuro-pneumonia has been observed in goats (Spinola, Koppitz, and others), camels, buffaloes, bison, and yaks. It is most likely that the reported cases of pleuro-pneumonia in pigs were founded on error. This remark also applies to the reputed cases in man, and especially in children. The fact that a form of pneumonia which occurs in children is anatomically related to pleuro-pneumonia, has no bearing on the subject. It is important to know that the flesh of animals which have died from pleuro-pneumonia can, as is the usual custom, be eaten by mankind without any injury to health.

Statistics of Epidemics.—1. In *Germany* during the last seventeen years (1886 to 1902) about 16,000 cattle (say 1,000 per annum) suffered from pleuro-pneumonia. The following figures show the great decrease in the frequency of the disease in Germany during that time: 1,778 (1886), 2,156 (1887), 1,545 (1888), 896 (1889), 528 (1890), 1,273 (1891), 1,182 (1892), 686 (1893), 822 (1894), 940 (1895), 1,608 (1896), 810 (1897), 672 (1898), 587 (1899), 468 (1900), 284 (1901), and 85 (1902). Contagious pleuro-pneumonia has, accordingly, declined from 2,000 in the year 1887 to 85 in 1902. The proportion of cases of the disease occurring in mid-Germany is as follows: 1887, 1,404 cases = 65 per cent. of all cases in Germany; 1888, 1,110 = 72 per cent.; 1889, 790 = 88 per cent.; 1890, 567 = 91 per cent.; 1901, 282 = 99 per cent.; 1902, 84 = 99 per cent. From these last figures it is shown, that even in the special disease foci, pleuro-pneumonia has declined from 1,400 to 80—that is to say, to about one-twentieth. Of the suspected cattle which were slaughtered from this central focus, from 60 to 70 per cent. proved to be affected with lung disease. Of 6,024 animals inoculated in *Russia*, during the years 1891 to 1893, 34 (= 0.5 per cent.) died in consequence of the inoculation sickness.

2. In *Prussia*, according to Felisch, 23,582 head of cattle died of, or were killed on account of, pleuro-pneumonia, during the ten years from 1876 to 1885. The figures for the respective years are as follows: In 1876, 3,117; in 1877, 1,980; in 1878, 5,098; in 1879, 2,364; in 1880, 1,749; in 1881, 1,982; in 1882, 2,079; in 1883, 3,070; in 1884, 3,252; and in 1885, 1,891. The total sum paid as indemnities within these ten years amounted to £225,000. These figures, compared with those of the last five years, show that the present laws have a restrictive effect on pleuro-pneumonia in Germany. The fact is particularly well proved by the statistics of earlier times. Thus, there occurred in Rhenish-Prussia, during the ten years from 1835 to 1844, no fewer than 100,000 cases of pleuro-pneumonia.

3. During the years 1890 to 1895 there were nearly 10,000 cases of pleuro-pneumonia in *Great Britain*;* the numbers being 2,471, 2,437, 1,843, 1,646, 1,466—total, 9,863. In the years 1897 and 1898, on the other hand, there was but one case of pleuro-pneumonia notified, so that England was free from the disease in 1899. In 1860, nearly 200,000 head of cattle succumbed in Great Britain; so that in a period of six years about 1,000,000 fell victims to the disease.

4. In *France* the number of cases for the years 1886 to 1890 were 1,480, 1,145, 1,289, 1,700, and 2,252, showing a total of 7,866. In the department of the Nord, which is one of the chief centres of the disease, over 200,000 head of cattle died of pleuro-pneumonia in 19 years. The departments of the Seine, and Meurthe and Moselle, which is a district in the south-east, are greatly affected by this disease.

5. In *Bohemia* and *Moravia* pleuro-pneumonia is very common. In 1888 and 1889 a total of 6,600 infected cattle were reported to the authorities. According to Röll 0·4 per cent. of all the cattle in these countries were affected from 1877 to 1887. The annual loss which occurred in Austria from pleuro-pneumonia, was estimated at £150,000.

6. In *Hungary*, 1,558 cattle were attacked during 1891 and 1892; in the years 1894 and 1895, 3,242; in 1899 and 1900, there were but 15 and 19 cattle nominally reported which the official authorities condemned as suffering from pleuro-pneumonia.

7. In *Belgium* there were 3,500 cases of pleuro-pneumonia from 1886 to 1890. On the other hand, the disease has not been met with for several years in *Holland*, where it was formerly constant in the Spöling district. In that centre, 600,000 cattle died of pleuro-pneumonia from 1830 to 1840.

The disease was introduced into *Australia* in 1858. During the years 1860 to 1872 one and a half millions of cattle suffered from it. At the present time it has spread over the entire continent, so that, for instance,

* The following statistics show the great success which has attended the system of stamping-out as regards bovine pleuro-pneumonia in England. In the year 1890, during the last third of which compulsory slaughter was introduced, 591 cattle were found to be infected; in 1891, 778; in 1892, 134; in 1893, 30; in 1894, 15; in 1895, 1; in 1896, 9; and in 1897, 46. Past experience and the restrictions on the importation of live stock give us the assurance that this terrible scourge which formerly devastated our herds, is now only of rare and sporadic occurrence. Austria, which in 1892 adopted the system of pole-axing all pleuro-pneumonia affected cattle and all cattle which had been exposed to its contagium, is now, like England, but little troubled by the disease.—TR.

the single colony of Queensland suffers a yearly loss of £640,000. In *America* about 10,000 cattle were attacked by the disease in the state of Illinois (Chicago) during 1886. In *Turkey* the disease appeared in the district of Adrianople in 1888. It has been reported that a disease resembling pleuro-pneumonia broke out among the ruminants of the nomad Kurds, and among buffaloes in Asia Minor.

Anatomical Conditions.—Pleuro-pneumonia is anatomically characterised by a progressive interstitial pneumonia with secondary hepatisation of the alveoli of the lungs, and spreading of the process to the pleuræ. Usually only one lung, the left as a rule, is infected. The anatomical changes vary according to the duration of the disease.

1. The otherwise healthy lung shows, in the initial stage, small, circumscribed, inflammatory centres the size of a hazelnut up to that of a walnut. The interlobular tissue in it is hyperæmic, permeated by single hæmorrhages and infiltrated by serum. The reddened lobules of the lungs are surrounded by bright margins, which are 0·04 to 0·08 inches broad, and which are filled with a serous or lymphatic fluid. When the deposits are superficial, the pleuræ become opaque and covered with slight clots.

2. At the height of the disease we find instead of the lobular inflammatory foci, a lobular pneumonia with pleuritis which is usually spread over the greater part of one lobe of the lung. The lung is considerably enlarged, of firm consistence, very heavy (weighing up to 1 cwt.), sinks in water, and does not crackle when cut into. Its section appears marbled, in consequence of the interstitial connective tissue having become thickened into broad lines, which vary in colour from orange to dirty-white, and which surround the darker-coloured lobules of the lungs. The larger lobules have a thickness of from 0·2 to 0·8 inches; and the smaller ones of from 0·1—0·2 inches. The colour of the enclosed lobules of the lungs depends on the duration of the process, and varies from brown-red to dirty-yellow. The recently infected lobules have a blood-red, reddish-brown, or dark brown colour (stage of red hepatisation). The colour of the older ones varies from orange to yellow (yellow hepatisation); and that of those of a still older date is grey (grey hepatisation). The central foci, because they are the oldest, are usually in the stage of yellow or grey hepatisation. Some of the inclosed lobules of the lungs are normal or only compressed, while others are merely hyperæmic. If we closely examine the bright interstitial lines, we shall find that they consist at first of

an œdematous infiltration, which, later on, becomes plasto-fibrinous, gelatinous, and indurated, and finally tends to the formation of adventitious connective tissue. The lymph-spaces in the lines are dilated like lacunæ and filled with a serous or fibrinous fluid. In robust animals, the exudate in the alveoli is croupy and firm; but is of a more serous character in animals of weak constitution. In the former case, a section made through the lung will be found to be granular. Besides these changes, the other lymph vessels of the lungs are dilated; their walls are infiltrated with cells, and their lumen is in a state of thrombosis. The blood-vessels frequently show thrombi and small hæmorrhagic infarcts. The contents of the finer bronchi are often fibrinous and infiltrated with numerous white corpuscles. The bronchial glands and frequently the mediastinal glands are inflamed and swollen.

The pleuræ are covered with soft, membranous, fibrinous masses, which are sometimes lumpy or crumbling, and which can easily be detached. These deposits have a reticular surface and may attain a thickness of 0·8 inches. If we remove them, we shall find the pleural vessels highly injected and suffering from well-developed ecchymoses, and the surface of the pleuræ in a rough and uneven condition. In the thoracic cavity we find, in varying quantity, a generally inodorous fluid exudate, which may be clear or opaque, and is infiltrated with flakes or lumps. Similar fibrinous masses often lie on the outer surface of the pericardium.

3. After the disease has been present for some time, we find in the affected parts of the lungs, induration, cicatrisation, caseation, calcification, necrosis, suppuration, etc. At first the interstitial infiltration becomes dense, solid and dry, and changes into firm connective tissue which makes a crunching noise * while it is being cut with the knife. In other places we have fatty degeneration, caseation, calcification, or suppuration, in which case the enclosed lobules of the lungs, in consequence of the existing suppuration, become gangrenous, and are cast off. They then form so-called sequestra, similar to those of bones, and are surrounded by a sequestrum cavity, which has a smooth wall. The dead portions of the lungs may remain unchanged in these cavities for a long time. Frequently they become softened to the consistency of a greasy yeast-like paste. Sometimes, when they are comparatively small, they get absorbed,

* Noise like that made when snow gives way under the pressure of the foot.—Tr.

and the cavity shrinks and forms a scar. The hepatised lobuli of the lungs rarely regain their normal condition after the absorption of the exudate. More frequently they atrophy or collapse, undergo atelectasis, carnification, caseation, calcification or softening, become necrotic or suppurating, or form cavities. On the pleuræ we find thick and wart-like hypertrophies of connective tissue, which frequently cause the lungs to adhere to the side of the chest.

The changes in the lungs and pleuræ, which we have already described, are the most important general changes in cases of pleuro-pneumonia. It is stated that we may sometimes meet with an interstitial fibrinous exudate in the liver with atrophy of the liver-cells; sero-fibrinous effusions into the articulations, tendon-sheaths, subcutis, dewlap and brisket; intestinal catarrh; areolation of Peyer's patches; and ulcers on the gastro-intestinal mucous membrane.

Symptoms.—The period of incubation varies on an average from 3 to 6 weeks, with a maximum of 16 weeks and a minimum of a few days. The course of pleuro-pneumonia is usually divided into a chronic and an acute stage.

1. The period of development (chronic or occult stage) is generally characterised by symptoms of chronic lung trouble, and lasts, as a rule, about a month, although in rare cases its duration may be restricted to only a few days. During that time the lungs show merely small, lobular centres of infection. The first symptom is a short, dry, painful cough, which, at its beginning, is so slight that it is hardly audible, and occurs more often in the early morning, after the animal gets up, and is drinking or moving about, than at other times. It gradually increases in frequency and intensity, so that it becomes exceedingly distressing to the patient, which spasmodically extends its head and neck and arches its back during the paroxysms. Feeding, rumination, and the secretion of milk are partly suppressed. The animal is slightly feverish, the internal temperature varying between 103° and 104° F. The external temperature of the body is unequally distributed. Percussion and auscultation continue to give normal results, or we may hear in some places vesicular respiration. On the other hand, the intercostal spaces are often very sensitive to pressure. In rare cases, the disease in this stage passes to recovery.

2. The open or acute stage progresses under symptoms of high fever, acute inflammation of the lungs and pleuræ, and

generally lasts 2 or 3 weeks; in rare cases, only a few days. The inflammation of the lungs spreads, and becomes lobular and diffuse. Respiration is very difficult and much accelerated. The animal breathes with widely-dilated nostrils and its flanks heave violently. It remains standing with the fore legs wide apart and the elbows turned out as much as possible, or lies down on the affected side for only a short time. The cough becomes sepulchral, and finally quite dull, and the ribs and vertebræ, especially behind the withers, are very sensitive to pressure. From the nostrils there is a mucous nasal discharge, which is sometimes mixed with blood and may become purulent and fœtid. Percussion produces at first a tympanitic, later on a dull, hollow sound, which extends over a considerable portion of the lungs, and has frequently a horizontal boundary. On auscultation we hear a feeble vesicular murmur, or, if it be not present, we hear in its place bronchial respiration, ronchi, and a friction sound. In the healthy lung, the vesicular respiration is much increased. The results of percussion and auscultation coincide with the well-known results of the physical examination which have been already discussed in connection with pneumonia and pleuritis. The temperature is from 104° to 108 F., and the rate of the pulse from 80 to 100 per minute, or more. The external temperature is very unevenly distributed. The ears and horns are sometimes warm, sometimes cold; the muzzle is dry and hot, the legs are cool, and the hair stands on end. Feeding, rumination, and the secretion of milk are entirely stopped, and thirst is increased. Usually constipation lasting several days sets in. In some cases, at the commencement of the second stage, we may observe diarrhœa with slight colicky pains, under the influence of which the animal moves about uneasily. The urine often contains large quantities of albumen. Pregnant animals sometimes abort. Finally, the patient becomes greatly emaciated. Œdematous swellings develop on the lower-lying regions of the body, as on the dewlap and on the lower part of the chest and on the legs. Breathing becomes difficult, pulsation frequent and weak, and the action of the heart palpitating. The patient, not being able to stand, lies groaning with outstretched neck, and at last dies from suffocation.

Progress and Prognosis.—Pleuro-pneumonia is sometimes acute, sometimes chronic. In young, strong animals which have been fed on good food, it generally runs a quicker and more acute

course than in old animals, or in those that have been kept on sloppy food for fattening purposes. In them the disease is slower, though more malignant. The mortality ranges from 30 to 50 per cent. Death usually takes place in from 2 to 4 weeks after the commencement of the acute stage. Occasionally, in severe cases, it may occur in from 5 to 8 days. In about 30 per cent. of all the cases, recovery is incomplete, in that chronic changes, with their consequences, remain in the lungs. The total loss consequently amounts to from 50 to 70 per cent. of all the infected animals, with a minimum mortality of at least 50 per cent. Complete recovery takes place in a smaller number of cases, in which improvement commences on about the fifth day of the acute stage. Convalescence is protracted for several weeks, as the absorption of the copious interstitial exudate goes on very slowly. In a few exceptional cases the course of the disease is abortive and the symptoms mild; there being only a slight cough, trifling acceleration in the breathing, and but little loss of appetite. The changes (necrosis, carnification, adhesion to the walls of the ribs, and new growths of connective tissue) that remain in the lungs after a typical case of the disease, cause, first of all, chronic troubles in respiration, with cough and emaciation, so that the aspect of the disease reminds one of tuberculosis of the lungs, with which malady pleuro-pneumonia is not unfrequently complicated. The disease in abortive cases has the peculiarity that relapses often occur after many months.

In large herds, at the beginning of an outbreak, only a few animals become sporadically infected; but the number gradually increases in a few weeks. In this way the disease continues to spread in the same shed for, say, from 3 to 6 months, and may finally become enzoötic in it. The intensity often varies greatly in different epizoötics. In previously attacked districts the disease always assumes a comparatively mild type. Insanitary stalls and bad food have a very unfavourable influence.

Diagnosis and Differential Diagnosis.—It is extremely difficult to form an exact diagnosis of the disease during its development; because the symptoms which are present are few in number, and by no means characteristic. The slight fever and cough are the only symptoms of diagnostic importance, when the suspicion of pleuro-pneumonia has been established to some extent. In the second or acute stage, a positive

diagnosis *intra vitam* can be made only when cases of pleuro-pneumonia had previously occurred, or when several occur simultaneously. As a rule, we can form a correct diagnosis only by a *post-mortem* examination. Bacteriological culture of the pleuro-pneumonic microbes, after preliminary filtration, now forms a means of diagnosis in the laboratory (see next page).

With regard to differential diagnosis, the following diseases come specially into consideration :

1. *Non-infectious croupy inflammation of the lungs*, which may be distinguished clinically, as a rule, by its more acute and more typical course, and by its sporadic and sudden appearance; and, anatomically, by the fact of the parts which have undergone hepatisation being all of the same age, and by the interstitial lung tissue and pleuræ being but slightly involved. Nocard states that in exceptional cases pleuro-pneumonia may run a very acute course and show hepatisation of equal ages.

2. *Tuberculosis* cannot be distinguished clinically, especially in the stage of development, from pleuro-pneumonia ; because in the former disease intermittent attacks of fever are also present in some cases. Tuberculin may be used experimentally as a diagnostic ; although it is not at all certain that animals which have suffered for some time from pleuro-pneumonia, do not react to tuberculin. An anatomical examination is the only real test. Tuberculosis and pleuro-pneumonia are frequently simultaneously present in the same animal.

3. *Traumatic pneumonia* or *pneumonia due to foreign bodies* may exhibit the same symptoms as pleuro-pneumonia. Evidence during life, of changes in the heart, caused by traumatic carditis, will be conclusive.

4. *The pectoral variety of bovine "deer and cattle disease"* may, under certain circumstances, show a great similarity to pleuro-pneumonia, on account of its epizoötic appearance. The differentiation is founded on the simultaneous occurrence of the exanthematic and abdominal forms of bovine "deer and cattle disease," and on the fact that this disease, as a rule, runs a rapid course. In it, the lesions of the lungs are of the same age, and the inflammatory process is much more acute than in pleuro-pneumonia (see Vol. II., p. 275 *et seq.*).

5. We can rarely make a mistake in *phthisis verminalis, simple catarrhal pneumonia, pneumonia due to foreign bodies, pneumomycosis, acute emphysema of the lungs, atelectasis of the lungs, actinomycosis, distomatosis of the lungs, mercurialism*, and other

diseases. The frequency of cases of fœtal atelectasis is an important practical point, which has been erroneously advanced against the unrefuted fact that the essential anatomical criterion of pleuro-pneumonia is the marbled condition of the lungs, which consists in an increase of breadth of the interstitial connective tissue, with hepatisation of the respiratory lung tissue of various ages, and consequently of different shades of colour.

Anatomical Differential Diagnosis.—According to Rabe, the anatomical diagnosis of pleuro-pneumonia can be affirmed only when, in addition to mortification of the lung tissues, the various inflammatory stages are also recognisable in other parts of the lung. Furthermore, the evidences of a pulmonary inflammation must be distinctly manifest in the mortified lung tissues (escape of red blood corpuscles into the alveoli). If the sequestrum consists merely of structureless, necrotic detritus, the diagnosis of pleuro-pneumonia is not justified. With regard to the various stages of the inflammatory processes, the alveolar tissues, the interlobular connective tissues, and the pulmonary pleura furnish sufficient proof. In the alveolar tissues, various changes are to be recognised. 1. A portion of the pulmonary lobule is yellowish-red in colour; the alveoli contain air, and a fluid rich in albumen. 2. Another portion is of a straw, varying to a citron, yellow tint; only an albuminous fluid is found in the alveoli; the cut surface is flat. 3. Another part displays the same general characters, but with a blood-red zone at the periphery. 4. Other pulmonary lobules are of similarly red colour, of somewhat firmer consistence, and present a cut surface, which is flat and juicy. 5. A number of lobules are in like manner tinged of a dark red colour, but of a liver-like density, and obviously granular in the cut surface. Of these changes, those of 3, 4, and 5, at least, must be simultaneously present. The interlobular connective tissue is, in 1 and 2, greatly expanded, pale yellow, gelatinous; the expanded lymphatics are filled with yellowish coagula. In 3 and 4, the consistence is somewhat firmer, the cut surface is homogeneous, and of a dull, glistening appearance; in 5, the connective tissue septa are paler, grayish-white and spotted. The pulmonary pleura is either distended with fluid, turbid, and coated with membranous exudations; or it displays a rind-like thickening from increase of the connective tissue, and is of a grayish-white colour.

Bacteriological Diagnosis.—For the purpose of isolating and demonstrating the microbes of pleuro-pneumonia, the serous fluid is collected from the lung, and filtered through a Chamberland bougie or a Berkefeld filter; which allows only the pleuro-pneumonic microbes to pass, but no other bacteria which may infect the lymph. From the filtered juice of the lung, a pure culture of the pleuro-pneumonic microbes may be grown in a few days, in Martin's peptonised broth, or on solid nutritive media (Agar).

Therapeutics.—The treatment can be only prophylactic and expectant; for the medicines which have up to the present been used, have had no curative effect in this disease. The best

results are obtained from the protective veterinary police measures laid down in the Imperial [German] laws with respect to epizoötics; the most important being that which orders the slaughter of all animals suffering from pleuro-pneumonia. This is the most certain means; for by it the infection is destroyed along with the infected animal. Strict disinfection with very strong agents should be carried out, and all tainted material burned. The disinfected shed should be left unoccupied as long as practicable. Some authorities recommend that it should not be used for at least a year and a half.

Special warranty is given against pleuro-pneumonia in Germany; the interval allowed, according to the new Municipal Code, being 28 days.

Septic Pleuro-Pneumonia in Calves.—Under this name, Pöls describes an epizoötic disease of calves which has a striking similarity to pleuro-pneumonia. The disease appears to be a form of septicæmia, accompanied by pleuritis and pneumonia. It runs a very acute course, with fever and great difficulty of breathing. The pathological changes in the lungs closely resemble those of bovine pleuro-pneumonia. Pöls states that the cause of the infection is a rod-like bacterium, which is very like the respective bacteria of contagious pneumonia of the pig, septicæmia of rabbits and "deer and cattle disease." Its pure cultures produce a disease very similar to contagious pneumonia when inoculated in pigs; and kill mice, rabbits, guinea-pigs, calves, and young horned cattle. We may easily distinguish Pöls' disease from pleuro-pneumonia by the microscopical demonstration of this bacterium.

Evers has observed various enzoötics of septic calf pneumonia in Mecklenburg, and thinks that he has discovered the bipolar bacteria of contagious swine pneumonia as the cause. The appearance of the sections was exactly that of contagious swine pneumonia. These were especially demonstrable in all stages in the chronic cases, from partial purulent and caseous pulmonary inflammation to complete necrosis. The treatment with the serum of contagious swine pneumonia (septicidin) also proved effective. The symptoms of the disease consist in depression, somnolency, frequent coughing, dyspnœa (40–70 respirations per minute), and high fever (104.9° to 106.8° F.). At the height of the lung disease, the calves stand with their fore-legs wide apart; the head and neck stretched out, the ears lying back; and show a laboured and painful respiration; diarrhœa appears a few hours before death. Prognosis is very unfavourable. Calves of from 8 to 14 days old usually die in from 2 to 4 days; older ones after 2 or 3 weeks. Evers has never seen a complete recovery. Schmidt-Kolding has also indicated the similarity of calf-pneumonia to many forms of contagious swine pneumonia; but leaves open the question of identity of the two diseases. As the infection takes place, as a rule, only through the air-passages, calf-pneumonia cannot be brought into any connection with calf diarrhœa (white scour).

Galtier found the cause, in calves, of an infectious pneumo-enteritis, which occurs particularly in Southern France, to be the *pneumo-bacillus*

septicus. This disease also occurs in lambs, sucking-pigs, and in a benign form in cattle, sheep, goats, and full-grown pigs, and can be transmitted by inoculation to these animals. The symptoms consist in bronchitis, pneumonia, pleuritis, inflammation of the intestines, pericarditis, and muscular degeneration. The mortality in this disease is very high. The fact of having successfully passed through one attack does not confer immunity. Beresow has observed in Russia an epizoötic pneumonia of calves with gastro-enteritis. There is lung hepatisation in foci, inflammation of the fourth stomach and intestines, ulcers in the abomasum, and swelling of the follicular mesenteric glands. Only calves of 8 weeks old and under became affected ; the majority being from 3 to 5 weeks old. The disease lasts for 3 or 4 weeks. The symptoms are : cough, muco-purulent nasal discharge, suppurative conjunctivitis, diarrhœa, emaciation and weakness.

Perroncito has described an infective pneumonia in calves, which caused great loss to farmers, and which is due, according to his researches, to the *micrococcus ambratus*. The colonies are nail-shaped, and pure cultivations on gelatine and agar-agar have an amber-yellow colour. The disease attacks calves up to the age of 3 months, and is said to occur also in sucking-pigs. Similar pulmonary inflammations in calves (appearing at the same time as contagious swine pneumonia), have been described by Semmer, Trinchera, Seiffert, Stöhr, Basz, Hürlimann, Imminger, Hutyra, Mehrdorf, Schick, Berg, Penberthy, Marder, Sauer, and others.

Preusze has observed an infective pneumonia in young pigs with symptoms resembling those of contagious swine pneumonia.

Infectious Pleuro-Pneumonia in Goats.—In 1894 there appeared simultaneously in several districts in Germany an epizoötic inflammation of the lungs, which had been introduced by Swiss goats from Saanen, and established itself among numerous goats in the Saxony Alps (Pusch and in Schmalkalden (Schütz). The symptoms of the disease, which broke out soon after the arrival of the goats from Simmenthal in Switzerland, consisted in cough, dyspnœa, painful condition of the thorax on percussion, mucous nasal discharge, orange discoloration of the mucous membranes, dulness on percussion, pleural friction sound, and great weakness. *Post-mortem* examination showed the presence of pneumonia and pleuritis. The disease was very infectious, and the period of incubation varied from 8 to 10 days. Some of the animals manifested a herpes-like eruption on the neighbourhood of the mouth. Pusch warns people against getting goats from Saanen in Switzerland. In Cape Colony, Hutcheon observed among goats an infective form of pleuro-pneumonia which had been introduced by Angora goats. The symptoms were those of serious chest trouble ; and the disease ended fatally in two-thirds of all the cases. Protective inoculation decreased the mortality by 30 per cent. The immunity thus conferred lasted 4 to 6 months. Similar cases have been observed : in France, by Jusquenoz ; and in Belgium, by Férir ; the latter cases may be regarded as true pleuro-pneumonia. Also, in Italy (Mazzini), in the Pyrenees (Leclainche), in Asia Minor (Nicole), and in the steppes of Kirghiz (Ben-Kiewitsch, Matwejen, Alexejew), similar goat-plagues have been recognised. The Kirghese people believe that camels also suffer from goat plague ("Kibeneck").

Epizootic Inflammation of the Lungs in American Oxen.—Nocard has described an infective broncho-pneumonia among American oxen which is produced by short, motile, ovoid bacteria that are found in the hepatised tissues, in the lymph spaces of the lungs, and in the exuded matter of the bronchi. Williams considers it to be a catarrhal broncho-pneumonia which is quite independent of pleuro-pneumonia. This disease is evidently identical with cornstalk disease or cornfodder disease, which Billings and others have described, and which has been observed for many years in the Western States of America among cattle that are pastured on stubble land. Here we have a specific septicæmia which is caused by an ovoid, very motile bacterium, and which, after it has lasted for some time, becomes almost always complicated by pleuro-pneumonia. This disease was regarded in England among imported animals as pleuro-pneumonia. It can be transmitted experimentally to calves and pigs (Billings, Spencer, Pyle, and Nocard).

Inoculation against Pleuro-Pneumonia.—Inoculation has been practised for a long time as the principal means for combating pleuro-pneumonia. As early as the beginning of the last century it was proposed in Germany by Haussmann and others. Its employment was greatly increased by the investigations which were made in 1851 by Willems in Holland, and which were published in 1852. Since that time these inoculations have been practised in nearly every country, and the literature on the subject has been very copious. In spite of all this, the question as to the value of inoculation as a protection against pleuro-pneumonia, has not yet received a final answer. The answer to this question depends especially on the consideration as to whether the object in view is the immunisation of individual cattle, or combating an epidemic, in a herd or in a locality. In this respect the following facts must be noted :

1. For the individual animal, a higher grade of immunisation is, as a rule, attained by inoculation.

2. On the other hand, the value of inoculation, as a veterinary police regulation in the campaign against pleuro-pneumonia, is still doubtful. Experience has frequently taught that all inoculated animals do not attain immunity ; but that about 2 per cent. of those operated on, remain susceptible to the infection in spite of the inoculation. These apparently immune cattle, after they contract the disease, in a latent form, and especially where there is a great cattle traffic, are a continuous source of infection for the healthy ones. Pleuro-pneumonia sometimes runs so mild a course, that from the absence of obvious symptoms of disease, the recognition of an outbreak of the epidemic, and the protective inoculation of the herd which

would have followed thereon, becomes altogether impossible. The long incubation stage of pleuro-pneumonia, also, often prevents the early establishment of a diagnosis; and subsequent inoculation. As a means of putting an end to the epidemic, the slaughter of the whole affected herd is much more reliable than inoculation.

The Advocates and the Opponents of Inoculation.—1. The advocates of inoculation, among whom we may mention Haubner, Pütz, Rueff, Bouley, Degive, Schütz, and Steffen, start from the well-known fact that one attack of pleuro-pneumonia successfully passed through, confers immunity for the remainder of the animal's life. By inoculation, a local, specific, inflammatory process, which is analogous to that in the lungs, is produced and is followed by subsequent immunity of the whole body. Inoculation also shortens the stay of the disease in a shed. The losses after caudal inoculation are stated to be insignificant. Haubner calculates that the mortality from inoculations is from 1 to 2 per cent., and that the tips of the tails are lost in from 5 to 10 per cent. of the cases. In Hol land, among 59,180 cattle inoculated in 1878 and '79, the mortality amounted to only 0.66 per cent. The experiments successfully made in Holland, Saxony, Anhalt, and Australia are cited as proofs of the benefits of inoculation. Further evidence on this point has recently been given by the positive results obtained by Schütz and Steffen. Dissemination of the affection by inoculation is denied. Inoculation has also been recommended as a far cheaper method than stamping out, which entails great pecuniary sacrifices. Degive has calculated that the results of inoculations undertaken in different countries from 1850 to 1883 show that 2.7 per cent. of 6,706 inoculated animals, and 26.9 per cent. of 2,453 non-inoculated beasts, became infected with pleuro-pneumonia, when they were exposed to the same pathogenic conditions as regards infection. According to Pütz, the number of cases of pleuro-pneumonia was reduced in Holland, in consequence of inoculation, from 6,079 in the year 1871 to 2,227 in the year 1875; 951 in the year 1877; 157 in the year 1879; and finally to 11 in the year 1882. It is also stated that from 1850 to 1880, in Hasselt, 200,000 head of cattle were inoculated with good results, and that the losses invariably rose when inoculation was neglected. Rochebrune mentions that the Moors in Senegambia have practised inoculation from time immemorial as a preventive against pleuro-pneumonia. They perform it by inserting the point of a knife into the lungs of a slaughtered animal, and then making with the knife an incision in the skin of the nasal region.

2. The opponents of inoculation, among whom we may mention the names of Roloff, Wehenkel, Lydtin, Œmler, Zündel, Kitt, Adam and McFadyean, assert that up to the present no positive case of immunity has been proved to have been obtained from inoculation. They also point to the fact that even the advocates for inoculation are unable to give the exact duration of the immunity, and consequently make several inoculations. The specific nature of the tumour produced by inoculation is disputed; for an exactly similar tumour appears after the inoculation of pus or milk. Again, inoculation never gives rise to the form of pneu-

monia met with in pleuro-pneumonia, which is the chief characteristic of the disease, and becomes developed when the disease is transmitted from a pregnant cow to the fœtus. Also, the results of inoculation are greatly influenced by the method and time of the inoculation, and by the quality of the inoculation material. It must frequently occur that immune animals are unintentionally inoculated, and that their immunity is credited to the effect of the inoculation. Inoculation would therefore cause the disease to be transmitted to other districts and to be artificially kept up. The opponents of inoculation state that the losses due to inoculation are often very considerable, and that the mortality sometimes exceeds that of pleuro-pneumonia. They further assert that in the documents upon which the German Imperial laws with regard to epizoötics are founded, the losses due to inoculation are stated to be from 2 to 4 per cent. A French commission puts the loss of the tips of the tails at 25 per cent.; Degive gives it at from 10 to 15 per cent. To this we must add loss from decreased yield of milk, emaciation, etc., as results of the inoculation. They also point out that the disease frequently spreads in spite of inoculation; that its progress sometimes becomes spontaneously arrested without inoculation; and that many animals pass through the disease unobserved. We must also bear in mind that in those countries in which inoculation has been most practised, the disease shows no decrease, as, for instance, in England,* where the official report lays particular stress on the doubtful value of obligatory inoculation for pleuro-pneumonia; and as in France and Belgium, in which countries inoculation is obligatory. In other countries, as in Holland, the decrease in the number of cases was not perhaps the consequence of the inoculation, but the simultaneous application of veterinary police measures, especially stamping out. Œmler, for instance, states that in Belgium pleuro-pneumonia increased in spite of inoculation from 1,481 cases in 1867 to 2,800 cases in 1878; but that it decreased rapidly in consequence of the application of strict veterinary police measures from 1,781 cases in 1880 to 1,187 cases in 1883. According to a table made by Kitt, the number of cases in England decreased only after the application of severe protective measures in 1878, during which year there were 4,590 cases. These numbers fell to 2,144 in 1879; and to 1,200 in 1882. Pleuro-pneumonia decreased in Bavaria, after the introduction of the German Imperial law respecting epizoötics, from 846 cases in 1846 to 281 cases in the year 1883. In Baden, where from 1870 to 1880 there was a yearly loss of 0.02 per cent. of all the cattle from pleuro-pneumonia, not a single case occurred in 1885.

It is difficult to draw a definite conclusion from the respective arguments of the advocates and opponents of inoculation. At any rate, inoculation seems to be useful, not as a protection, but as a means of shortening the duration of the epizoötic in a large stationary herd of affected cattle. We certainly regard slaughtering the diseased animals as the best means for combating pleuro-pneumonia, the infection of which, being unable to exist independently of the animal body, can be annihilated along with it. Stamping out, strict disinfection of the tainted sheds, and their evacuation for a long period, consequently offer a much better chance of rooting out the disease than inoculation.

* *See* foot-note, p. 491.—Tr.

Methods of Inoculation.—The tail is the part usually inoculated, chiefly by the following methods :—

1. *The old method.*—In order to obtain the material for the inoculation, an animal which is in the first stage of the disease is slaughtered, and the affected parts are cut out of the fresh lungs. By gentle pressure exerted on the removed portions of the lungs, we obtain lymph, which we should allow to coagulate, and should then filter it through a piece of clean linen. Others use as lymph the fluid which is obtained by incisions made into the inoculation tumour on the tail, and which is said to have a comparatively mild effect. The site of the inoculation is on the dorsal aspect of the tail and about 3 to 4 inches from the tip. After removing the hair, the inoculation is made at one or two spots with a hypodermic syringe, which should be made to penetrate the subcutis. The lymph should be clear, of the colour of yellow hock, and should never be taken from necrotic foci in the lungs, but only from those parts which are in the stage of hepatisation. The inoculation " takes," on an average, in from 75 to 90 per cent. of the inoculated animals. After a period varying from 1 to 4 weeks, there appears on the site of the inoculation an inflammatory swelling, which, under normal conditions, is not larger than a hen's egg, and which is accompanied by slight fever and accelerated breathing. If the lymph is not quite pure, extensive swelling of the whole tail with necrosis of the tip, high fever, and symptoms of septicæmia and pyæmia, will probably appear. These symptoms are also observed when the inoculation is made on the dewlap or at the root of the tail, on which account these places must be avoided for the inoculation. If the tail becomes excessively inflamed, we must prevent an unfavourable result by free incisions, antiseptic treatment, and, if need be, by amputation of the necrotic tip. As already mentioned, the mortality is on an average from 1 to 3 per cent., and the loss of the tips of the tails, from 5 to 15 per cent. Sometimes a second inoculation is made after 6 or 8 weeks on a place situated above the site of the first inoculation. Martin inoculates the tail immediately under the last caudal vertebra by passing a seton 0.11 inch broad and saturated with lymph. Rutherford obtained favourable results by this method, with a mortality of 2 per cent. Bad consequences are said to rarely follow this method. The swelling caused by the inoculation becomes developed between the ninth and the fourteenth day ; lasts for 7 or 9 days ; and disappears in from 14 to 20 days.

2. *Pasteur's method.*—A healthy calf, 2 or 3 months old, is inoculated subcutaneously with ordinary good pleuro-pneumonia lymph on the dewlap or behind the shoulder. After the animal has been killed, the lymph is sucked up by means of sterilised glass tubes of small calibre inserted into the large swelling which has become developed in the neighbourhood of the inoculated part. The collecting tubes are half filled and are then quickly closed at both ends by means of a spirit flame. Whilst the lymph is being sucked up, the free end of the tube should be closed with a stopper of wadding, to prevent the entrance of air. In these tubes, which can be sent anywhere, the lymph keeps in an attenuated form for 6 or 8 weeks. It is stated that a few drops of this lymph will suffice for the protective inoculation of a full-grown beast. By inoculating a calf from time to time, fresh lymph can always be obtained. This inoculation with so-called secondary calf-lymph has, according to the experiences of the last years in Prussia, not proved satisfactory

(Wanzleben). The immunity which is established by preliminary inoculation with secondary calf-lymph on the tail does not afford sufficient protection against the powerful effect in cattle of a second inoculation on the dewlap with a strongly virulent lymph (Jarotschin). On this account the older methods of inoculation (lung-lymph, tail-inoculation) have again been prescribed in Prussia.

3. *Schutz's and Steffen's method.*—The lungs are entirely taken out of the thoracic cavity and are incised on the affected part to a depth of 0.4 inch by means of a sterilised knife. The parts are torn asunder by the disinfected hands, and the lymph is sucked up by sterilised syringes. The site of the inoculation being on the posterior surface of the end of the tail, the hair immediately above the tuft of the tail is clipped for a width of 4 inches, shaved off, and the part washed with soap and water, and moistened with a solution of sublimate (1 to 1,000), or of creolin (1 to 20). The point of the needle is introduced through the skin obliquely inwards and downwards, and the contents of the syringe are injected into the subcutis. The punctures made by the needle are closed with cotton wool which has been dipped in sublimate collodion (1 to 1,000), and the cotton wool is then fixed with a strip of adhesive plaster 19½ inches long and 0.4 inch broad. The subsequent local inflammation resembles that of erysipelas. Sometimes a portion of the tail, or the whole of it, becomes necrotic and is cast off. The local reaction is more violent after the injection of warm lymph than after that of cold lymph.

4. *Method of Nocard.*—Lung-lymph is replaced by cultures of the microbes of pleuro-pneumonia. The effect of the cultures is more lasting, milder, and also more certain than that of the lymph. Of 970 cattle so inoculated none died; in 3 only, loss of the tail resulted. Serum-inoculation has not hitherto proved satisfactory.

5. *Intravenous injections.*—Besides caudal inoculations, Thiernesse, Defays, Bouley, Degive, Sanderson, and others, have tried injections of ½ fluid dram of the lymph into the jugular vein. The results are said to be very good and more certain than those of caudal inoculation. It is said to have given rise in one case to a marbled inflammation of the lungs.

DOURINE (*Maladie de Coït* or *Covering Disease*).

Dourine in horses—Transmission of human syphilis to animals by inoculation.

History.—This disease was first observed by Ammon in the Trakehnen stud in 1796. In the year 1817 it appeared in Hanover in the stud farm of Celle and in Prussia, where it caused great losses during the thirties of last century, especially in Silesia and Poland, so that in 1840 a law containing a number of protective measures against it had to be passed. From the year 1821 we find this epizoötic in Austria, especially in Bohemia and

Hungary, where it was widely disseminated at times, as for instance, from 1859 to 1862 in Bohemia. In 1830 it was introduced into Switzerland and France, where it also caused very great losses among the horses of the breeding establishments; in 1836 into Italy; in 1843 into Russia; and later on into Algiers, Syria, and other countries. According to Röll it is unknown in England and Belgium. Although it was very prevalent for some years in Germany, it has almost entirely disappeared since the introduction of the German Imperial laws with regard to epizoötics. For several years not one case has been mentioned in the statistics of the German states. In Hungary, 69 horses were affected with this disease in the year 1898.

Various views have been held respecting the nature of this disease. It has been commonly supposed to be identical with human syphilis, as is evident from its old names—"equine syphilis" and "chancre plague." Even in recent times it was considered to be syphilis transmitted from man to mares, which view was held in France by Bouley, Trasbot, Laguerrière, and others. The above-mentioned authors assumed that the disease was transmitted at first by syphilitic Arabs to she-asses, and from them to he-asses and mares. Laguerrière therefore proposed the name of "*syphilis des équidés*" for this disease, which is known in France as *Maladie de coït*, or *dourine*. This theory of origin is unsupported by any proof. Others state that they have seen several cases of glanders and farcy spring from dourine, and consequently they regard it as identical with glanders of the genital organs. This mistake is evidently due to the fact that in a few exceptional cases, true genital glanders was mistaken for dourine. For a considerable time, dourine was described by some authors as an independent disease of the spinal cord, the principal symptoms of which were stated to be paralysis. It was called on that account "paralytic disease" (Strauss), "nerve disease" and "breeding paralysis," which is a name that is frequently applied to it at the present day.

The fact that two entirely different diseases, namely, dourine and vesicular exanthema, have been for a long time described under the one name of "covering disease," greatly contributed to the confusion which existed respecting the nature of this malady. The old literature on the subject contains many descriptions in which the symptoms of the two diseases are hopelessly mixed up. Only in recent times has it been found necessary to distinguish between a benign and malignant form of dourine, namely, between true dourine and vesicular exanthema,

the true character of which was first recognised by Hertwig. Even at the present day we require more exact and more complete clinical, bacteriological and anatomical information concerning the etiology and symptoms of the two diseases, especially of dourine, which we shall discuss first. More recent investigations of this disease have been carried on by von Thanhoffer, Marek, Schneider, and Buffard.

Etiology.—The dourine of horses is, according to the results of French investigators (Schneider and Buffard), produced by a flagellate animalcule (trypanosoma equiperdum), which is similar to the trypanosoma of the surra disease; and that of the tsetse-fly disease; but is, however, smaller and more slender, and possesses the faculty of migrating through intact mucous membranes. This trypanosome is found as a very motile, thread-shaped, filaria-like parasite, of 20–30 μ in length, and 1.5–2 μ in breadth; in the blood corpuscles of the blood-stained serous fluid of the fresh pimples; and also in the semen, but seldom in the circulating blood of horses affected with dourine. The demonstration of the trypanosome is not always easy, as it disappears temporarily from the fluids named. They may be inoculated—by all the inoculation methods—on the horse, ass, dog, rabbit and mouse; infection follows most rapidly on intraperitoneal and intravenous injection. The results which most closely correspond to the natural course of the disease develop from subcutaneous inoculation. Infection sometimes occurs, also, when a few drops of blood containing the trypanosomes are placed on the conjunctiva or the vaginal mucous membrane. The stage of incubation varies, in cases of inoculation, between 4 and 20 days.

Dourine is a chronic infective disease which is at first localised on the mucous membranes of the genital organs, and from thence produces a general infection of the body. It is exclusively transmitted through infection during the act of coition from stallions to mares, and *vice versâ*. Consequently, it is chiefly disseminated during the covering season, especially by stallions, and less frequently by mares. One stallion may, for instance, infect all the mares covered by him. On account of the chronic course of the disease, the infective matter may remain apparently latent in the system of a horse for a very long time, even for more than a year. The disease, as Hertwig was the first to show, can be transmitted by inoculation with the secretion

of the genital mucous membranes. Nocard has found that inoculations with the contents of the centres of softening in the spinal cord are successful in conveying the disease. Inoculations made by Trasbot and St. Cyr with blood, semen, prostatic fluid, and transfusions of blood gave negative results. Nocard has succeeded in producing the disease in dogs by inoculations into the anterior chamber of the eye, and found that they died after from 6 to 11 weeks with symptoms of paralysis. According to Rodloff, Jessen and others, the disease is hereditary.

Dourine, Surra and Tsetse-Fly Disease (Nagana).—Besides dourine, the Indian surra and the African nagana or tsetse-fly disease are also produced by trypanosomes (see Vol. II., p. 646). Dourine is, however, not identical with these diseases as was formerly thought (Nocard). The trypanosomes of dourine are smaller and more slender in contrast to the tsetse-fly disease (fly-puncture), the infection usually takes place only from the act of copulation; the course is chronic (surra and nagana display a rapid course); finally, ruminants are immune to dourine while they are highly susceptible to the other two diseases (tsetse-fly disease).

Marek has recently thrown doubt, on the grounds of negative microscopic researches and inoculation results, on the etiological relations of the trypanosome to dourine. He holds that it has not been determined; that the trypanosomes are accidental conditions; or that the Algerian "dourine," which is produced by trypanosomes, may possibly be no disease of copulation, but only a similar one in appearance. De Docstras. in the Dutch Indian Settlements, found trypanosomes but twice among 10 cases of dourine in horses.

Anatomical Conditions.—The changes in the genital organs consist in an œdematous or phlegmonous swelling of the vulva and its neighbourhood in females; and of the penis, sheath and scrotum in males. After the disease has continued for some time, the skin of the sheath frequently becomes indurated and considerably thickened. Catarrhal swelling and thickening, ulcers, and scars may be seen on the mucous membrane of the *labia pudendi* of the vagina and of the urethra, and on the outer surface of the penis. The ulcers may be superficial or deep; and the cicatrices smooth and white, or firm and elevated. The mucous membrane of the vagina often shows circumscribed, pustular or villous hypertrophies, by which the calibre of the vagina becomes contracted. On the vulva and penis we also find unpigmented spots, corresponding to the former seats of the ulcers. The testicles are

either enlarged or decreased in size and contain caseous centres of inflammation. Between the *tunica vaginalis* and *propria* we find adhesions of connective tissue and villous proliferations. The connective tissue of the epididymis and spermatic cord is often infiltrated by a yellow gelatinous exudate. The udder is sometimes inflamed, swollen, and filled with abscesses.

The lymph glands in the neighbourhood of the genital organs are often swollen, pigmented and permeated with caceous foci the size of a pea up to that of a nut. In the abdominal cavity, the subperitoneal tissue is also gelatinously infiltrated, and the mesenteric glands may become swollen to the size of an apple. In one case Ruthe observed rounded ulcers on the intestinal mucous membrane. We find on the skin, in greater or less number and in various sizes, circumscribed, flatly-raised wheals (like those of urticaria), which are somewhat hard to the touch, and represent a serous inflammation of the cutis (vaso-neurosis). The respiratory mucous membrane undergoes a catarrhal change in some places. The cadavers are excessively emaciated and anæmic. In the lungs we sometimes find a hypostatic pneumonia or metastatic centres, if pyæmia has been present.

Condition of the Nervous System.—According to the most recent researches made by Marek, the nervous disturbances, which manifest themselves during the course of dourine, are caused by a polyneuritis of the peripheral nerves ; he therefore calls this disease polyneuritis infectiosa equorum. Marek says that the central nervous system (the brain and spinal cord) remains perfectly intact in dourine, the posterior columns of the spinal cord alone showing slight changes. On the other hand, most of the peripheral nerves, and more especially the nervous ischiaticus, n. peroneus, n. tibialis and n. cruralis, disclose symptoms of neuritis in the form of degeneration, round-cell infiltration, increase of the endothelial cells, and sometimes also a new formation of connective tissue. This polyneuritis is caused by the poisoning of the blood by the infectious matter created in dourine, and the toxins which this produces. They primarily irritate the connective tissue (neuritis interstitialis), and a secondary degeneration of the actual nerve-parenchyma ensues. A cellular infiltration with contained blood vessels may also develop in other organs besides the peripheral nerves (genitals, muscles, spinal cord).

Earlier Researches.—In the spinal cord we find, according to Thanhoffer, symptoms of myelomycosis, which manifests itself as a *myelitis hæmorrhagica centralis*, as a syringo-myelitis (a central medullary sclerosis), or as a localised degenerative process. At more or less numerous places the spinal cord is attenuated; and its transverse sections are asymmetrical, softened, and permeated with red spots. The grey matter is atrophied, and contains pulpy centres of softening of greater or less size. The spinal meninges are opaque, adhesive, hyperæmic, and have a considerable quantity of serous fluid between them. The microscopical examination of the spinal cord reveals abnormal changes in the ganglionic cells namely, in the nucleus and body of these cells, axis cylinder processes, neuroglia (which is considerably increased), blood-vessels, etc. The membranes of the brain, especially the pia mater of the cerebrum, are hyperæmic and opaque. The ventricles of the brain contain a considerable quantity of serous fluid, and the brain substance is sometimes very œdematous. The inter-fibrillar connective tissue and the neurilemma of the larger nerves which come from the spinal cord, are gelatinously infiltrated.

Symptoms.—The disease is characterised by local inflammation of, and by the formation of ulcers on, the mucous membrane of the genital organs, with subsequent general derangement, chiefly manifested by spinal paralysis, and vaso-neurosis of the skin (urticaria).

1. The primary local changes in the genital organs begin after a period of incubation, of, according to Maresch, from 8 days up to 2 months. The first symptom in the stallion consists in swelling of the penis, especially of the glans penis, which, later on, may become considerably swollen and parephimosis may take place, similar to what sometimes happens after castration. On the outer surface of the penis we may see red spots, vesicles and ulcers. The *meatus urinarius* is reddened and swollen and shows a mucous discharge. The animal has a continuous desire to mictu ate and frequently manifests sexual excitement. The swelling also spreads from the penis to the sheath and scrotum, in which case the testicles become inflamed (*orchitis*). Finally, the inguinal glands and lymph vessels become inflamed. These local affections may, in the further progress of the disease, almost entirely disappear. In many cases nothing can be seen of the external changes just described, because the mucous membrane of the urethra is first of all affected; the only visible symptoms being strangury and, on closer examination, a mucous discharge from the urethra.

In mares the disease begins with a doughy or tense swelling of the pudenda, which frequently spreads to the udder and inner surface of the thighs. The mucous membrane of the

vagina is red in spots and swollen, sometimes thickened by gelatinous elevations, and covered with a turbid orange-coloured secretion. We may sometimes observe on the mucous membrane nodules, vesicles, and ulcers, which, however, are frequently absent. Also in mares the local changes may often be insignificant. The mucous membrane in the neighbourhood of the clitoris is more congested than at other parts, and the clitoris itself is swollen and erect. At the same time, affected mares show excessive sexual excitement. They frequently suffer from strangury, and, after considerable straining, discharge urine in jets, or in small quantities, or a little sticky discoloured mucus. They incessantly shake their tails, are very ticklish, and open and close the vagina in rapid succession, while showing the clitoris in the manner common to mares in season. The discharge often assumes a corrosive ichorous condition, so that the tail and the hind legs become soiled and even scalded. In severe cases, the neighbouring lymph glands are inflamed and swollen as well as the udder, on which abscesses may appear. The swelling may even extend to the hypogastrium.

2. The general symptoms develop only after several weeks or even months; in fact, their appearance is often delayed until the local symptoms have disappeared. At first the animals are depressed and weak; they frequently continue to lift up their hind feet alternately, so as to try to avoid putting weight on them; knuckle over on their fetlock joints; lose control over the movements of their hind legs while walking; and often drag a hind leg.

Especially stallions suffer from an attack of urticaria, in the form of sharply-defined, round, flat eminences, which may be raised the breadth of a finger above the surface, and may vary in size from a shilling up to a half-crown, or even be larger. These eminences are caused by a serous infiltration of the papillary layer of the skin in the neighbourhood of a small artery, and are evidently of a vaso-neurotic character. They often appear and disappear rapidly and may shift their position. Usually they persist for several weeks, during which time they become moderately hard, and then slowly disappear. Their favourite sites are the croup, neck, shoulders, chest, and belly.

During the further course of the disease, a progressive paralysis of the hind quarters, combined with excessive emaciation, sets in. The animal has a staggering gait; often gives way on the pasterns and knees; knuckles over behind; can raise itself from the ground only with difficulty; and sometimes falls down

unexpectedly. The affected stallion is unable to cover; for he can neither mount a mare nor get an erection. Some patients exhibit permanent tremblings over the whole body, or local paralysis, as for instance, that of the lips, ears, and eyelids (*ptosis*). We may also observe, particularly in stallions, a striking hyperæsthesia of the skin, which manifests itself in the form of an intense pruritus, so that the animal continually rubs itself, bites the affected parts, and thus produces extensive sores on the skin. The patient becomes extremely emaciated, especially in the hind quarters, so that the outlines of the pelvic bones and ribs are very prominent. The skin is dry, and the coat stands on end and loses its gloss. Some of the animals manifest pain when the lumbar region is pressed. The sensorium becomes more and more dull and blunted; and the eyes assume a staring and expressionless appearance. Towards the end the sufferer persistently maintains a recumbent position, and finally perishes from hypostatic inflammation of the lungs, septicæmia induced by the decubitus, or general cachexia. Sometimes in the final stage the patient suffers from nasal catarrh with swelling of the submaxillary glands, and conjunctivitis. Laguerrière has observed severe internal inflammation of the eyes. The appetite continues normal longer than any other function.

Course and Prognosis.—The course of dourine is always chronic; its average duration being from 6 months to a year, although it may extend to 4 years or even longer. In very few instances the disease runs an abortive course, in which case the morbid changes are merely local. Relapses sometimes occur, after a considerable improvement has been noticed. The local symptoms frequently disappear when paralysis sets in.

The prognosis is unfavourable, as the mortality amounts to about 70 per cent., and is more unfavourable in stallions than in mares. Recovery is possible only at the beginning of the attack.

Marek observed an enzoötic of dourine in Hungary in 1898 (70 mares affected in 8 districts of a county), and on that occasion studied the disease thoroughly, in both its anatomical and clinical aspects. He states that dourine begins, in most cases, with disease of the genital organs; while, in other instances, the changes in these organs are so slight in degree that they escape observation. The first symptom of disease of the nervous system is offered by the typical round, flat eminences (affection of the vaso-motor nerves). These present a circumscribed hyperæmia of the skin, with serous infiltration of the tissues. Besides these, cutaneous eruptions like urticaria appear, also swellings of the joints. Simul-

taneously, or somewhat later, symptoms appear, which indicate lesions of the sensory nerve fibres; namely, hyperæsthesia of the skin and itchiness, with formation of vesicles and swelling of the skin (herpes ?); painfulness of the peripheral sensory nerves on pressure, and also on movement (nervous ischiadicus, n. peroneus, n. medianus, n. infraorbitalis, n. intercostalis); painful drawing up of the hind legs in the stable; acceleration of the cutaneous reflexes (withers, thoracic, abdominal, gluteal, perineal and anal); and diminution of the patellar and muscular reflexes. Finally, there also appear, as a result of disease of the motor fibres, peripheral motor paralyses, with a very changeable aspect of the disease.

In ischiatic (sciatic) paralysis the corresponding hind-foot when walking strikes the ground with the wall of the hoof. In peroneal paralysis the animals stumble and knuckle over when weight is placed on their backs. In tibial and crural paralysis there are abnormal flexions of the corresponding joints at the time of bearing weight. In ischiatic paralysis also, stringhalt is seen, and in radial paralysis the animals stumble and brush the ground with the outer surface of the hoofs. In facial paralysis, there is unilateral paralysis of the face, and in paralysis of the oculomotor and abducens, squinting of the eyes and ptosis. An analogy with the tabes dorsalis of man (degeneration of the posterior columns of the spinal cord) does not exist, from the peripheral nature of dourine. In contrast to the tabes dorsalis of human beings, it is through the neurotic nature of dourine that a possibility of cure is offered.

Differential Diagnosis.—Before the urticaria and spinal paralysis manifest themselves, it is often very difficult to diagnose the presence of dourine on account of its chronic and exceedingly slow course. The diagnosis is more difficult in the stallion than in the mare; because the external local changes are frequently not visible in him, in which case the disease can be recognised only by the fact of the stallion infecting several mares.

Dourine is most easily mistaken for vesicular exanthema, which is an acute and usually entirely benign exanthema of the genital organs with local changes very similar to those of dourine, but without any general disturbance. Formerly, severe cases of vesicular erythema have been frequently diagnosed as dourine. The inflammation and formation of ulcers on the vaginal mucous membrane may also be mistaken for glanders, especially when the lymph vessels and lymph glands are simultaneously inflamed. In this case the further progress of the disease and inoculation experiments will enable us to decide the point. Phlegmonous inflammation and pyæmic processes with swelling of the lymph glands, as may be sometimes observed after coition in mares and stallions which are suffering from an ichorous purulent discharge,

may be mistaken for dourine. Here, also, the further progress of the disease will decide the matter. Chronic paralysis of the loins may be mistaken for the second stage of dourine.

Therapeutics.—Treatment can at most be successful only in the first stages of the disease, and then only by treating the local lesions with disinfectants, such as solutions, respectively, of corrosive sublimate, carbolic acid, etc. Later on, the local application of disinfectants and astringents (solutions of lead, alum, tannin, sulphate of iron, etc.) can have only a palliative effect. The external swellings of the skin, udder, and lymph glands should be treated by incisions—cantharides ointment, carbolic ointment, etc. Internally, the usual remedies for human syphilis, for instance, corrosive sublimate $1\frac{1}{2}$ to 3 grs., potassium iodide $\frac{1}{3}$ to $\frac{2}{3}$ oz., and arsenic 7 to 15 grs., have, it is said, been used successfully. The spinal paralysis may be treated with strychnine and irritant cutaneous inunctions. It is stated that the disease has been frequently cured in stallions by castration; the supposed rationale of the operation being that, as the infection was contained in the testicles, their removal would include that of the virus.

On the Possibility of Transmitting Human Syphilis to Animals by Inoculation.—In spite of a great number of inoculation experiments a final answer to this question cannot be given with our present state of knowledge. It is stated that syphilis has been transmitted to monkeys, and the following positive results are claimed :—

1. In rabbits (Auzias-Turenne, Gailleton, Waller, Klebs, and others).
2. In guinea-pigs (Legros, Bradley, Michot, and others).
3. In cats (Auzias-Turenne, Bradley, and Diday).
4. In dogs (Auzias-Turenne and others).
5. In horses (Mathieu and others).
6. In pigs (Matineau, Hammonic, and others).

Against these positive results we must place a large number of negative ones obtained by Hering, Blaiso, Lesing in horses ; Horand and Peuch in dogs, cats, and mules ; by Hunter, Ricord, Horand, Cornevin, and Teleschinski in pigs ; by Werthheim, Jessen, and Tissoni in rabbits, dogs, and cats ; by Hunter, Depaul, and Reynal in cattle ; by Lacroix and Danet in cattle and rabbits ; by Rebatel in dogs, guinea-pigs, and rabbits ; by Letnick in pigs and rabbits ; Mazyck P. Ravenel in the calf ; Brieger and Uhlenhuth in goats, fowls, and guinea-pigs. Fournier also utterly denies the possibility of inoculation of syphilis on animals.

Syphilis of animals has only an experimental pathological interest ; because its transmission, under ordinary circumstances, from man to any other animal has never been observed. We have never been able,

especially with dogs, to accept as positively proved a case of the transmission of syphilis to one of the lower animals, in spite of various statements and suppositions. There occurs just as little in domestic animals, an infective catarrh of the mucous membrane of the urethra, namely, true gonorrhœa (clap of man); the clap of dogs being merely a purulent catarrh of the prepuce, in which the mucous membrane of the urethra does not participate. The transference of human gonococci on experimental animals has not hitherto succeeded.

VESICULAR EXANTHEMA OF HORSES AND CATTLE.

1.—*Vesicular Exanthema in Horses.*

Etiology.—This vesicular eruption in horses is a generally benign and typically progressing, infective vesicular exanthema of the mucous membrane of the vagina and skin of the vulva and penis. We place it under the present heading only because of its veterinary police relations; for it really belongs to the acute exanthemata. It is met with in many animals, most frequently in horses and cattle, and rarely in sheep, goats, and pigs. The nature of the infective matter is not precisely known. The disease can be transmitted with the greatest facility, as we ourselves have observed in our inoculation experiments. The usual mode of infection is by coition; but cases are not at all rare of mares becoming infected by other mares, without previous coition. Even foals may become infected, during sucking, on the lips, mouth, mucous membrane of the nose and eyes, and on the hind quarters (Dayot, Lundberg, Hess, and others). The infective matter is found only in the contents of the vesicles and pustules and in the secretions of the ulcers. The disease is frequently epizoötic; for one stallion may infect a large number of mares. A previous attack does not confer immunity; in fact, stallions which have recovered from the disease often become again infected after a few weeks (Steinhoff). The eruption is sometimes transmitted to man, especially to the hands of attendants, in a form which resembles small-pox, and may lead to a swelling of the entire arm, and axillary glands, and to a feverish increase of temperature. The patient, however, always makes a rapid recovery. The vesicular exanthema is consequently looked upon in France as a kind of horse-pox (Trasbot). Inoculations in cows and calves are said to produce characteristic cow-pox (Peuch and Galtier).

Symptoms.—1. The period of incubation varies from 3 to 6 days; but is often only from 12 to 24 hours. In ordinary mild cases in mares there appears, first of all, on the inner surface of the labia pudendi and on the greatly reddened mucous membrane of the vagina, dark red spots, the size of the head of a pin, which soon change into nodules, vesicles and pustules, the size of a lentil up to that of a pea. These vesicles may at first be transparent, and in their further progress may change to various shades of orange. They have a very thin covering membrane, and are found more numerously in the neighbourhood of the clitoris than on the outer surface of the swollen vulva, or on the under surface of the tail, where they also appear. The vesicles develop later on into flat ulcers which have a deeply reddened base, and which secrete a sticky, lymphatic, yellowish fluid. These ulcers often become confluent, and covered with a brown scab. On healing, they leave behind round, smooth, white cicatrices. Frequently there is present, at the same time, eczema, great sexual excitement, desire to micturate, and more or less intense vaginitis. There are no symptoms of fever or other constitutional disturbance.

In stallions the penis is swollen, externally reddened, and in some places, especially in the cervix of the penis, it is covered with pimples, vesicles and pustules, which subsequently form ulcers of greater or less depth. These ulcers on healing leave an unpigmented cicatrix. In rare cases, the eczema spreads to the outer surface of the sheath and scrotum. In one instance, Schleg observed the eruption on the inner surface of the thighs and fore-legs, and on the throat, which had probably become infected by contact with the secretion of an infected mare during coitus. There is also a discharge from the urethra; and the animals frequently stretch themselves out to stale and draw their yards. The eruption will usually heal spontaneously in mares and stallions after 3 or 4 weeks without the application of any remedies.

2. In severe cases the swelling of the pudenda of mares spreads anteriorly to the udder and even as far as the chest, and posteriorly to the hind legs as far as the hocks. The lymph vessels and the lymph glands become attacked and suppurate; and abscesses form in the udder, about the anus, and near the root of the tail. The ulcers become very deep, give off an ichorous discharge, and sometimes extend into the uterus. We may observe at the same time grave constitutional disturbance, high fever, stiff gait and considerable emaciation, accompanied, in

a few cases, by death (Steinhoff). The disease may last for 6 months, or even longer. A chronic vaginitis frequently remains after the inflammatory swelling has subsided. In stallions the disease may run a tedious course with swelling and abscesses of the inguinal glands, fever, digestive disturbances, and lameness.

These severe cases of the disease were formerly the frequent cause of vesicular exanthema having been mistaken for dourine, on account, perhaps, of septic or pyæmic complications having been present. The treatment in such cases is the same as for dourine (antiseptic treatment, incisions, etc.). The benign variety of vesicular exanthema may be treated by disinfectant and astringent solutions, such as those of carbolic acid, creolin, lysol, corrosive sublimate, alum, etc.

Statistics of Epidemics.—The statistics of the last 13 years (1886 to 1898) show that in Germany, at least, the vesicular exanthema of horses is about 20 times as rare as that of cattle. Both, however, agree in the fact that they are most frequently observed in spring, namely, during the covering season. We cannot possibly prove that centres of this disease, as in other epizootics, exist in Germany. It seems, however, that during the last few years frequent cases of vesicular exanthema of cattle have been observed in the Black Forest, in Schleswig, Saxe-Weimar and Meiningen, Cassel, Wiesbaden, and Middle and Lower Franconia. During the last 13 years there occurred in Germany altogether about 3,000 cases of vesicular exanthema in horses, and 80,000 cases in cattle. The respective years show the following figures :—

Year.	Horses.	Cattle.
1886	248	3,944
1887	178	5,233
1888	200	6,874
1889	115	5,224
1890	267	5,782
1891	305	4,739
1892	145	4,969
1893	83	5,299
1894	151	8,147
1895	329	6,232
1896	336	9,523
1897	230	8,140
1898	329	6,751
1899	174	5,911
1900	167	6,132
1901	166	6,710
1902	227	8,898
	3,650	108,508

2.—Vesicular Exanthema of Cattle.

Etiology.—This disease, the nature of the virus of which still remains unknown, is very infectious. Frequently, all the cows of a village become infected by the same bull. Cases of transmission, independently of coition, occur more commonly than in horses. Cows often infect each other by intermediate bearers, especially when close together. For instance, the eruption has been transmitted to cows that were at the end of their pregnancy by sponges with which infected cows had been cleansed. Dinter saw the infection communicated by one cow to 9 others by the rubbing together of their hind quarters and by the whisking of their tails. Numann saw the eruption in the neighbourhood of the anus and sheath even in oxen. According to Armbruster, the infective matter is frequently transmitted to healthy cows by these animals lying with their hind quarters against infected wooden troughs. In 1885 Fenner saw the infection in 669 non-pregnant cows, and also in cows far advanced in pregnancy, and in calves of 5 weeks old. Kampmann maintains that the infection can be transmitted by the air. Schnieper, Fenner and others state that cattle may be attacked several times. One bull, for instance, was 3 times infected. Trasbot asserts that vesicular exanthema in cattle is simply transmitted horse-pox.

Symptoms.—The average period of incubation in cows varies from 1 to 6 days, with a maximum of 10 days. The first symptom is excessive vaginitis, the mucous membrane of the vulva and vagina is greatly congested, swollen, covered with dark red points or spots, and gives off a mucous discharge resembling the white of egg. It also develops translucent yellowish vesicles and pustules the size of a grain of millet up to that of a pea. The pustules change into ulcers, at which stage the mucous discharge becomes purulent, and dries upon the thighs and tail in the form of hard crusts. Frequently the ulcers are fairly deep and have jagged wall-like edges and a dirty base. On healing, they leave white scars, some of which are smooth; others
discharge, The swollen vulva is painful to the touch, and the
observe at t ers are very sensitive and irritable. Considerable
fever, stiff frequently present, so that the animals continually

rub themselves and whisk their tails. They often move in a very stiff manner, and suffer from strangury and sexual excitement.

If numerous ulcers are present, the discharge becomes very abundant, and assumes an ichorous and corrosive character, in consequence of which the neighbourhood of the vulva and the skin of the hind legs become excoriated. In a few severe cases we find necrotic lesions the size of a walnut up to that of a hen's egg, on the mucous membrane, when, for instance, an injury of the mucous membrane was present prior to the infection. As a rule, the temperature is only temporarily raised to a slight extent; but the appetite, rumination, and secretion of milk are more or less suppressed. The disease rarely causes abortion. Chronic catarrh of the vagina and permanent sterility are frequent after-consequences of this disease.

In bulls the glans penis, sheath, and scrotum are often inflamed, swollen, and painful to the touch. Pimples, vesicles, and ulcers, the size of a hemp seed up to that of a pea, form on the penis, especially on the glans, and a thin, yellowish, mucopurulent discharge flows from the urethra. Frequently strangury and phimosis are present. During erection, hæmorrhage sometimes takes place from the ulcers. The animal loses its appetite, is dull, and suffers from slight constipation and suppressed rumination. In exceptional cases extensive gangrene of the penis may take place, with permanent deformity of that organ (Kampmann).

Usually the disease lasts only for a week or two, although it may extend to a month in severe cases. The assertion lately made that the disease may assume a chronic form, lasting for several months, with granular hypertrophy of the vaginal mucous membrane, has been proved to be erroneous; for granular (follicular) processes on the vaginal mucous membrane, quite unconnected with this disease, may occur in infectious vaginal catarrh (colpitis follicularis infectiosa) as well (see next page); and even may, as experience proves, also occur in very many otherwise healthy cows. This disease, indeed, has lately been often confused with infectious vaginal catarrh (compare Differential Diagnosis, Vol. II., p. 521). All those chronic granular or follicular vaginal inflammations are distinguished from this disease, not only by their chronic course, but by the absence of vesicles and pustules. On the other hand, the affection may frequently continue for from 2 to 4 months in a shed. This complaint may be mistaken for foot-and-mouth

disease, variola, acute and chronic vaginitis, and perhaps also for rinderpest. The treatment should be expectant. In severe cases, disinfection and astringent lotions (solutions in water of creolin, carbolic acid, alum, sulphate of copper, sulphate of iron, etc.) may be employed.

Infectious Sexual Disease in Rabbits.—In a superior breed of rabbits we have observed a very infectious inflammation of the external genital organs (vulva and prepuce) which was capable of being transmitted from one sex to the other, and was distinguished by a considerable swelling of the parts and an abundant muco-purulent secretion. Treatment with a 1 to 2 per cent. solution of sulphate of copper, continued for several weeks, cured the disease.

INFECTIOUS VAGINAL CATARRH OF CATTLE.

(*Colpitis follicularis infectiosa.*)

Causes.—The infectious vaginal catarrh of cows ("nodular eruption of the vagina," "vaginitis verrucosa," "colpitis granularis"), which is widely distributed throughout Germany, but especially in Thuringia, is characterised by a prominent swelling of the lymph follicles of the vaginal mucous membrane (colpitis follicularis). It is caused by a specific streptococcus (Ostertag). The disease is excessively infectious. The infection chiefly occurs through bulls which have served affected cows. The communication of the infective matter also takes place from one cow to another by means of the straw, dust, and utensils of the cow-shed. It thus affects, not only pregnant cows, but also heifers, and even calves. The disease is not communicable to horses, sheep, goats, pigs and dogs.

Symptoms.—The disease begins with the symptoms of an acute purulent vaginitis: redness, swelling, pain, and a muco-purulent coating of the vaginal mucous membrane, with swelling of the vulva. After some days, there appear on the swollen mucous membrane of the vaginal vestibule, especially on the lateral surfaces and in the neighbourhood of the clitoris, numerous flat, dense nodules (swellings of the lymph-follicles), in size varying from half a millet-seed to half a hemp-seed; and at first of a dark red, and afterwards of a bright red colour. At the same time there appears an inodorous, purulent or muco-purulent vaginal discharge, which dries on the vulvar hairs and on the upper surface of the tail to dirty brown crusts. The general condition of the animals is not greatly affected.

After from 3 to 4 weeks, the acute inflammatory redness and swelling of the vaginal mucous membrane subsides, the discharge becomes mucous, the swollen lymph-follicles dwindle; but remain visible for about a month, in the form of bright red to yellowish red nodules. The economic consequences of this infective vaginal catarrh consist of sterility and abortion of the diseased cows, as the result of extension of the catarrh to the uterus (endometritis).

Differential Diagnosis.—The vesicular eruption is, in contrast to the infective vaginal catarrh, an acute, benign lesion, in which no nodules, but vesicles, pustules and ulcers appear, and heal in a short time, leaving a smooth surface. The general condition of the animal is affected in the vesicular eruption. Bulls also sicken in a remarkable manner, and develop vesicles and ulcers on the sheath and penis, with a muco-purulent discharge from the sheath. Finally, the vesicular exanthema also occurs on horses, sheep, goats and pigs.

Treatment.—Infective vaginal catarrh is a lesion which is difficult to cure. Among the remedies which have proved most effective are: douching of the vagina with creolin, lysol, bacillol (2½ per cent.), with subsequent tamponade of the vagina (0.1 per cent. ichthargan solution). The disease is curable in from 2 to 3 weeks by this method. At the same time, the neighbourhood of the vulva, and the entire shed, are to be thoroughly disinfected with creolin, lysol, or bacillol (3 per cent.) (Raebiger). Others recommend insufflation of pulverised alum, alum-creolin (97 : 3), or alum-toxic acid (4 : 1).

Enzoötic Vaginal Necrosis in Cows.—Ellinger has described an infectious vaginal necrosis, produced by the necrosis bacillus, and sometimes appearing among cows, as a generalised disorder at the calving season. The disease seems to stand in casual relationship with panaritium and foot-rot; and is to be regarded as a puerperal infection, which may lead to a fatal issue, with the symptoms of septicæmia. In combating its progress, the most important part is played by prophylaxis (separation, disinfection of the vulva and vagina before calving, disinfection of the shed.)

ANTHRAX.

Anthrax in general—Anthrax in cattle—Anthrax in horses—Anthrax in sheep and goats—Anthrax in pigs—Anthrax in dogs and cats—Anthrax in birds—Anthrax in man.

A.—ANTHRAX IN GENERAL.

History.—Anthrax is perhaps the oldest - known infective disease of animals. It appears to be alluded to in the Second Book of Moses, ix. 10, as the sixth plague of Egypt. In the Third Book of Moses stress is laid on the possibility of transmitting the disease by clothes to man. The epidemic which was described by Homer in the First Book of the *Iliad*, and which raged amongst men, mules, and dogs, seems to be anthrax. Ovid has given in the Ninth Book of his Metamorphoses a tolerably exact description of an outbreak of anthrax. Plutarch reports that Rome was visited about 740 B.C. with a severe epizoötic of anthrax. Dionysius of Halicarnassus (488 B.C.) and Livy (425 B.C.) relate examples of epizoötics of anthrax in which the disease attacked, at first, cattle on pasture, then those in sheds, sacrificial animals, priests, herdsmen, country people, and finally the entire population. In Lucrecius (428 B.C.) we find for the first time the name of "ignis sacer" applied to anthrax; and in Columella, the name of "pustula." Virgil describes an infectious ovine disease which was transmitted to man in consequence of wearing the hides or wool of stricken animals, and which produced deep pustules on the skin. Pliny mentions a disease of boils in the province of Narbonne, in Gaul, at the time of the migration of nations. The Arab physicians described anthrax as "Persian fire." Mezeray (966 A.D.) was the first to use the name "Ignis St. Antonii." Johannes Wierus describes in the second half of the sixteenth century several epizoötics in Italy (1552, 1598, and 1599), during which periods the Senate in Venice forbade the sale of beef on pain of death. Athanasius Kirchner describes in 1617 a bovine disease which infected mankind so that 60,000 people died of it. Anthrax of the tongue spread to an extraordinary extent during 1662 in the neighbourhood of Lyons, and throughout France during 1710 and 1731. Ramazzini states that anthrax of the throat raged in 1690 in Padua amongst oxen and pigs. Anthrax appeared during 1712 in Germany (at first in the neighbourhood of Augsburg) and in Hungary; during 1726, in Poland, Silesia,

and Saxony; during 1731 and 1757, in France as gloss anthrax, or carbuncle of the tongue, among almost all the domestic animals (horses, donkeys, cattle, sheep, pigs, deer, dogs, fowl, fish) and men. In 1755 and 1761 it appeared in Franconia; in 1758 and 1759 in Finland and Russia; in 1774 in Guadaloupe (West Indies). Chabert demonstrated in 1780 that the different kinds of anthrax were really one and the same disease. He classified and named them in a manner which has been followed up to the present day. Kausch published in 1805 a good description of anthrax, but denied its contagiousness. Since then, we may mention, in particular, the invasions of anthrax in the years 1807, 1810, 1819, and 1827, from which time a gradual decrease of the disease has taken place. Delafond and Gerlach (1845) investigated ovine anthrax in a very careful manner. Although the contagiousness of the disease was not recognised by Delafond, it was experimentally proved by Gerlach. Heusinger published in 1850 a comprehensive treatise on anthrax, especially from an historical and geographical point of view. He regarded anthrax as a malaria neurosa.

The knowledge of anthrax was greatly advanced about the middle of the last century. In 1855, Pollender, of Wipperfürth, published the discovery, which he had made as early as 1849, that an innumerable number of extremely fine rods existed in the blood of anthrax-stricken cattle. These rods were independently observed in 1850 by Davaine (Paris), and in 1857 by Brauell (Dorpat). Brauell found the rods in the blood during life, and used them as a diagnostic and prognostic factor; but he denied that they represented the virus of anthrax. It was only in 1863 that Davaine declared that they were the cause of anthrax, and were bacteria, against which theory Bouley, Sanson, and other authorities, at once protested. Cohn was the first to call these rods bacilli, and to suspect the existence of permanent spores. To R. Koch is due the credit of having demonstrated the development of these permanent spores from the rods and the transformation of the spores to bacilli. He also made pure cultivations of the bacilli, and clearly explained the biology of the bacillus anthracis. Protective inoculation against anthrax, which was introduced by Toussaint, and was widely advanced by Pasteur and others, is also of great scientific and practical interest.

Bacteriology.—Anthrax is an infective disease that is caused by a bacterium, the bacillus anthracis, which belongs

to the rod-like schizomycetes, and is specially characterised by producing resisting spores.

1. The *bacilli of anthrax* present themselves under the microscope in various forms, according to the methods used in preparing and staining the preparations. In fresh, unstained drops of blood they form limpid, non-motile rods with rounded ends, and may then be easily mistaken for other bacilli (the bacilli of putrefaction). In stained cover glass preparations we see the bacilli from 1·5 to 2 μ long, and from 1 to 1·5 μ broad, attached to one another, mostly in the form of a chain, which is from 5 to 20 μ long. The straight, slightly bent, or kinked chain-rods show a very characteristic method of division; as there are present at the place of union of the individual bacilli, transverse, fine, colourless luminæ by which the individual links seem to be transversely cut off. The ends of the individual bacilli in the chains assume various forms according to the method of staining. They appear to be connected sometimes in a straight line, and at other times in a bi-convex or bi-concave series, like the segments of a bamboo cane. Lately, special importance has been ascribed to the presence of an area which can be but very little, if at all, stained, or of a capsule-like covering, or membrane, in the neighbourhood of the cell-body of the individual bacilli. The bacilli of anthrax are to be found in all parts of the body, especially in the internal organs. They increase in the living body by transverse division and longitudinal growth (asporogenous bacilli).

Outside the animal body the bacilli form long filaments, which are often twisted and intertwined in various ways. They never ramify and may become 100 times as long as the original rod. The contents of the threads gradually become finely granulated, and there appear in it in regular order highly refractive and sharply defined oval bodies, namely, the spores, which become free after the disintegration of the threads (sporogenous bacilli).

The bacilli of anthrax can be cultivated on different nutrient materials, such as upon and in gelatine, in fluid nutrient substrata, on potatoes, agar-agar, etc. Surface cultures on gelatine form dirty-white, cloudy opacities, which liquefy gelatine. When we use a weak magnifying power, we find the cultivation to be composed of a confused mass of threads, which pass over the edge of the colony and return, and resemble whipcord. Stab cultures in gelatine show, in the track of the needle, a dirty-white line, which gives off radiating branches or thin sprouts,

and consists apparently of a fine crystalline structure (bacilli); and the gelatine liquefies. Potato cultivations produce a pale, dirty-white covering, in which the rods bear oval spores. On agar-agar they form white, bluish, and slightly-shining cultivations; and in fluid nutrient substrata (blood serum), grey, flocculent layers which lie on the bottom of the tube.

The bacilli are stained very easily with basic aniline colours, especially with an alkaline solution of methylene blue; but the spores do not take this stain. The bacilli stain by Gram's method.

The Biological Relations of the Bacilli of Anthrax are not less important than the morphological relations already described. Here we have to specially consider the influence of certain nutrient materials, of oxygen, of temperature, and of certain antiseptic and disinfectant substances.

(*a*) The following *nutrient mediums* may be mentioned: blood, blood plasma, blood serum, transuded matters of the body, aqueous humour, milk, and other mammalian secretions and excretions, animal substances and excretions which are mixed in the soil, especially alkaline fæces of cattle, neutral or slightly alkaline meat broth, meat peptone gelatine, raw and boiled potatoes, slightly alkaline infusions of hay, infusions of many grasses, with the addition of prepared chalk, which neutralises their acids, infusions of pea-straw, juice of carrots, turnips, swedes, beetroot and potatoes, and crushed cereals, particularly wheat and legumes. A certain amount of water, which must be neither too large nor too small, is necessary for the development of the bacilli. A larger proportion of water than 1 to 20 hinders their development. Thus the bacilli die in a day in distilled water and in cool spring water (Hochstetter). They perish in the ordinary temperature of a room or of an incubator (Meade Bolton), in water which is considerably mixed with organic matter, and at the temperature of well water (Wolffhügel and Riedel). The bacilli, in thin layers of tissue and blood, are destroyed by drying in from 12 to 30 hours; in moderately thick layers, in 2 or 3 weeks; and in very thick layers, in 4 or 5 weeks.

(*b*) The *oxygen* of the air is indispensably necessary for the development of the bacilli of anthrax, as they are ærobic. For this reason, spores never develop in the interior of an anthrax cadaver which does not contain sufficient oxygen for that purpose (Koch). Johne states that inside the flesh of a skinned and cut up animal, neither filaments nor spores develop, even if the flesh is exposed for several days to a rather high summer temperature. For the same reason, anthrax blood enclosed in tubes loses its power of infection after 8 days. On the other hand, spores and bacilli form on the surface of the flesh of animals which have died of anthrax, and which have been cut up into joints, if from the beginning there has been a sufficiency of moisture and warmth. Boschetti found very virulent bacilli after 8 months in meat prepared in this manner, and Savarese found them in a sausage which had been made of anthrax flesh 4 months previously. According to Semmer, spores also form in the internal organs. Klett thinks that the spore-formation is not

dependent on the presence of oxygen in the air ; it proceeds much more vigorously in an atmosphere of nitrogen.

(*c*) Temperature is a very important condition in the development of the bacilli, the most favourable being one of 95° F. Temperatures above and below that standard either retard the action or nullify the virulence of the anthrax bacilli, the development of which ceases entirely at 53° F., and also if the bacilli are buried deeply in the ground ; because the temperature of the ground is always under 53° F. Development is completely arrested at 113° F., and death takes place under the prolonged influence of a temperature of 131° F. For the preparation of his inoculation material, Pasteur has taken advantage of the fact that exposure to high degrees of temperature decreases the virulence of these microbes. With this object he cultivated for a long time the bacilli under oxygen at 108° to 110° F. Toussaint produces his inoculation material by warming the bacilli up to a temperature of from 122° to 131° F. The bacilli are killed by cold only in very rare cases. According to Feser, the cold of winter does not affect their vitality unless it falls under 14° F. and continues as low as that for at least 3 days. Gibier prepared attenuated inoculation material by cooling the bacilli down to − 49° F. Klepzow states that a three days' exposure to a temperature of − 14° F. does not destroy the bacilli.

(*d*) Arloing states that *light*, especially sunlight, impedes the growth of the bacillus.

(*e*) The bacillus of anthrax is destroyed by *putrefaction*. According to Bongert, the bacilli remain active in decomposing blood for from 8 to 20 days (on the other hand, when it has been dried, for from 36 to 50 days). When the discharges and detritus containing bacilli from the bodies of victims of anthrax stagnate on impervious floors, the bacilli perish under the influence of the factors of decomposition, so that spore-formation does not occur. Feeding experiments have shown that it is killed by normal gastric juice. Even spore-containing bacilli are killed by gastric juice in half an hour (Straus and Wurtz).

(*f*) *Chemical agents* influence the bacilli in various ways, either by checking their development or by killing them. Thus, development is stopped by corrosive sublimate (1 to 300,000—1,000,000), mustard oil (1 to 33,000), potassium arsenicate (1 to 10,000), iodine (1 to 5,000), bromine (1 to 1,500), salicylic acid (1 to 1,500), carbolic acid (1 to 1,000), boric acid (1 to 800), quinine (1 to 600), etc. The bacilli are killed by corrosive sublimate (1 to 30,000), formaldehyde (1 to 20,000), creolin (1 to 15,000), carbolic acid (1 to 100–200), thymol and salicylic acid (3 to 1,000), permanganate of potash (1 to 1,000), sulphurous acid, alcohol, etc. On the other hand, the bacilli appear to be unaffected by either the external or internal employment of iodoform. Forster states that the bacilli die in from 18 to 24 hours if common salt is sprinkled on them, and in less than 18 hours when the spleen, blood, etc., of animals affected with anthrax are salted. He also says that the spores retain their vitality after having been salted for months. The same remark applies to brine for pickling meat (Abel).

2. The *spores of anthrax* are formed only outside the animal body, by the free ends of the bacilli becoming continually elon-

gated. The protoplasm of the filaments which have by this time sprung up, becomes granulated and the spores make their appearance for the first time in the form of very minute and highly refractive granules, which gradually enlarge into true oval spores. The bacilli are produced from the spores by the spores becoming elongated in the direction of their long axis towards one side in the form of an oval, cylindrical process. This process gradually grows into a bacillus, and the spore becomes disintegrated and finally disappears. If from an infected animal the spores get into the soil, they may remain there in a state of arrested development for many years; ten or even more. The spores, quite independently of the animal body, may, however, vegetate in the soil and surface water, which fact is of great practical importance; for it explains the manner in which the disease becomes enzoötic in certain districts, in which the contagium may retain for many years its capability of becoming developed without the importation of fresh cases being necessary for its appearance. We also see how the disease may spread in any direction by the instrumentality of feeding materials, soil, flowing water, inundations, etc. The anthrax bacillus is consequently not only an endogenous, but is also an ectogenous bacterium. Under the latter condition it produces a miasmatic; and under the former, a contagio-miasmatic infective disease.

[Observation of the enzoötic manner in which anthrax occurs in certain tropical and semi-tropical parts of the world, such as Eastern Bengal, Manipur, and Burma, in certain years and seasons, among different kinds of animals, as for instance, horses, cattle, and buffaloes, apparently without the agency of infection of one animal to another, strongly supports the theory that, under favourable conditions of soil and climate, anthrax bacilli can maintain a vigorous ectogenous existence. In countries like England, on the contrary, their life outside the animal body is probably that of exotics.—TR.]

The biological relations of the spores of anthrax differ from those of the bacilli. Oxygen and a certain degree of heat are also necessary for the formation of spores; the most favourable temperature being 95° F. The temperature limits are 53° and 109° F. An abundant formation of spores takes place as early as 2 days with a temperature varying from 68° to 77° F., and consequently under the influence of summer heat, or in the ordinary temperature of a dwelling room. According to Arloing, light (especially that of the sun in summer, and even in November), (Ward) impedes and even entirely arrests the development of both bacilli and spores. The spores offer much more resistance to various injurious

influences than the bacilli. Thus they remain alive in water for an exceedingly long time, and for many years when in a dried condition. They can successfully bear exposure to 230° F. for 10 minutes or to − 166° F. for several hours. Putrefaction takes a long time to kill them. The best disinfecting agents for destroying the spores are corrosive sublimate, creolin, chlorine, bromine, and iodine. They are killed by a 1 to 1,000 solution of corrosive sublimate in 10 minutes; by a 2 per cent. watery solution of chlorine, bromine, or iodine in 1 day; and by a 3 per cent. solution of creolin in 48 hours (Eisenberg). It is also stated that formaldehyde (1 to 1,000) kills them in an hour. The spores remain virulent after having been placed for 20 days in a 5 per cent. solution of creosote; for 19 days in a 10 per cent. solution of lysol; or for 12 days in a 5 per cent. solution of carbolic acid (Lignières). Also by the correct process of tanning of the skin, the spores found on it are not destroyed (Griglio). According to Weil, the spore-formation in anthrax bacilli does not take place gradually, but suddenly; at 64.4° F. in 70 hours; at 75.2° F. in 16; at 98.6° F. in 8 hours. The process of development is arrested by the presence of carbolic acid (1.5 per cent.), and by formalin (1 per cent.).

General Pathogenesis of Anthrax.—It is very improbable that the bacilli and spores of anthrax ever pass directly from one animal to another. Anthrax is not a directly infectious disease; although the virus may adhere to any part of the body, especially by means of secretions and excretions. When the transmission of anthrax from one animal to another occurs, it is almost always effected by intermediate bearers, such as utensils, people, and insects. The great majority of cases of anthrax is caused by a miasmatic infection from the soil. Here we must recognise a three-fold mode of immigration of the infective matter, namely, by the alimentary canal, by the skin and the natural openings of the body, and by the lungs. In cattle, the bacteria are usually received by the alimentary canal; and in horses and sheep by the skin, or by the intestines.

1. *Infection of the intestines*, which is the usual form, comprehends those cases of anthrax to which the names of "intestinal anthrax," "fodder anthrax," "spontaneous anthrax," "internal anthrax," "anthrax without external manifestations," "anthrax without localisation," "anthrax fever," etc., have been given. The spores, and in less number the bacilli, are received by the animal from the food and drinking water; the chief port of infection being the small intestine, the mucous membrane of which need not necessarily be injured. Although the acid gastric juice, as in tuberculosis, kills most of the bacilli, it is unable to injuriously affect the free spores. The principal

vehicles for the spores in intestinal anthrax are those feeding materials which have been grown on or near places in which animals suffering from anthrax have died, or have been superficially buried, or have deposited their excrements; and fodder obtained from infected districts. Some of the spores which are contained in the surface of the soil may become, by rain or dust, attached to various food-plants, or some of the infected soil may adhere to them, as for instance, to potatoes, turnips, etc. Water which carries the spores into the intestinal canal may, like fodder, be taken from infected places, such as wells, swamps, bogs, ponds, puddles, etc., which were contaminated by carcases of infected animals, tanneries, wool-washing establishments, etc. The infection may also be imported in artificial manures, unburnt bone powder, hair manure from tanneries, blood manure, etc. Carnivora may become infected by feeding on the carcases of diseased animals; and sucking animals, by infected milk. The spores may be introduced by the renewal of the flooring of the stall or of the litter by, respectively, infected earth or infected bedding. Pasteur's supposition that the spores were brought by earth-worms, which, according to Darwin, bring up soil to the surface from below, and thus cause a constant renewal and mixing of the superficial crust of the earth, was declared by Koch to be untenable; because, as he said, the temperature of the ground at a depth of from 1 foot 8 inches to 3 feet 4 inches was too low for the formation of spores, and because it was experimentally demonstrated that the contents of the intestines of worms which had been kept in anthrax soil were proved by inoculation to be non-poisonous. Bollinger has, however, experimentally proved the correctness of Pasteur's view. Five per cent. of the earth-worms of an anthrax pasture sent to him was found to contain the contagium of anthrax. As Karlinski, Proust, and others have shown, the spores of anthrax may also be disseminated by slugs and even by insects and larvæ which are found on untanned, infected skins (*dermestes vulpinus*), and which excrete anthrax spores in their fæces.

2. *Infection of the skin* is the cause of those cases of anthrax to the local manifestations of which, "carbuncle disease" and other names have been given. This mode of infection is comparatively rare and appears only sporadically. In such cases, the bacilli and spores penetrate through wounds in the skin and mucous membranes of the openings of the body, while

the animals are grazing, and by means of infected utensils, bites of shepherds' dogs, blood-letting, infected instruments in operations, skins (even tanned skins), insects, especially the *musca domestica* and *vomitoria*, and various kinds of gad-flies and simuliæ. Bollinger and Zeilinger succeeded in producing anthrax in rabbits by inoculating them with flies which had been caught on an anthrax cadaver. Anthrax has been experimentally produced by rubbing cultivations of anthrax into the skin of guinea-pigs (Machnoff).

3. *Inhalation-anthrax* is the rarest form. In it the spores penetrate through the lungs into the body. The severe pulmonary symptoms which present themselves in sheep are possibly referable to inhalation-anthrax (depressed position of the head during transport). It has been experimentally proved that healthy organs of breathing can be infected in this manner (Feser, Buchner, Lembe, and Enderlen). According to the researches of Galtier, infection from the nasal mucous membrane and from the conjunctiva, is also possible. Snel considers that the anthrax bacilli perish in healthy lungs, and on intact mucous membranes.

Besides these 3 kinds of infection, it seems possible that the disease may be transmitted from the mother to the fœtus (placental infection). Latis, for instance, succeeded 8 times out of 15 cases in transmitting anthrax to the fœtus through the mother in guinea-pigs. Malvos and Rosenblath believe that their inoculation experiments prove the transmissibility of the bacilli of anthrax from the mother to the fœtus. Although the placenta is normally impermeable to bodily elements, it becomes patent under the injurious influence of the anthrax bacilli, which, if we may use the expression, grow through from the maternal side into the fœtal side of the placenta (Birch-Hirschfeld). The fœtus has not been found to contain bacilli in all cases of anthrax (Fambach, Schaller, etc.).

The dissemination of the spores and bacilli in the body takes place in anthrax of the skin, first of all, by local increase of the bacilli in the skin, and subcutaneous connective tissue. The dissemination in the firm cutis is slow ; the bacilli migrating, according to Siedamgrotzky, in the skin at the daily rate of only about 0·2 inch. Consequently, from a cutaneous infection of inoculation, only a carbuncle arises ; although from a subcutaneous or submucous infection the bacilli can spread rapidly and produce the so-called œdema of anthrax. The further dissemination in the body takes place chiefly by the lymphatic

and blood-vessels. It is somewhat peculiar that the anthrax bacilli can be found in the blood only a very short time before death. It seems when the anthrax bacilli first penetrate from the local infected part into the blood, they fail to increase in it to any marked extent on account of healthy blood having an unfavourable action on them. Consequently they disappear from it and become deposited in the internal organs, especially in the spleen, lungs, and liver, where they freely multiply. It is only after the anthrax vegetation has made a certain amount of progress in these internal organs that the blood loses its germicidal properties and becomes a good nutrient material for the bacilli, which then increase enormously in that fluid (Frank and Lubarsch). According to the experiments of Rodet, the bacilli are to be found in the blood before the first hour after inoculation. The bacilli are most numerous in the capillary regions of the internal organs, as in the spleen, intestinal mucous membrane, mesentery, mediastinum, and lungs. For this reason, one often fails to find them in the blood of the peripheral and larger vessels. The enormous accumulation of bacilli in the capillaries frequently leads to laceration of these vessels and to extravasation of blood. Metastases of large accumulations of bacilli produce carbuncles and œdema in the internal organs and skin, accompanied by gelatinous extravasations and immigration of white blood corpuscles.

The effect of the bacilli in the blood is a subject about which little is known. Bollinger's mechanical theory of capillary engorgement and the theory of the deprivation of oxygen certainly explain the occurrence of hæmorrhages, but not the other features of the disease. The mechanical theory was directly opposed to the chemical theory, which asserted that the products of the metabolism of the bacilli, and not the bacilli themselves, were the cause of the symptoms of anthrax. Many objections have been raised against this chemical theory. Klebs, Pasteur, Nencki, and others, having separated the bacilli from their nutrient fluid by means of filtration through porcelain and with the aid of an air pump, have always found that the fluid itself was not virulent. In no case has anthrax been produced in animals by the inoculation of blood, or of liver or spleen tissue, or of material obtained from the fœtus of a mother whichwas suffering from anthrax. In more recent times, however, Hoffa prepared nearly pure products of anthrax bacilli, and found these products to be extremely virulent, chemical, basic bodies which, as he states, produced in animals the symptoms of anthrax. He terms them anthrax-alkaloids (*Anthracin*) and considers that they are allied to the *neurin* of Brieger's ptomaines. Hoffa considers that the poisonous action of these anthrax-alkaloids and not the abstraction of oxygen from the blood by the bacilli, or the mechanical blocking up of the capillaries, is the true cause of death.

The effect of the virus of anthrax on the animal body seems to be

southern localities between the Isar and the Inn (Weilheim, Erding).

In Prussia, anthrax is found more especially in the provinces of Saxony and Thuringia. The Sangerhaus district and the two Mansfeld districts are also known to be continuously affected. These lands, which are inundated every year, are formed of a dark, loose soil, rich in mould, containing lime-stone, and having a gravelly substratum; partly, too, of a moorland basis. In years of drought, much foul, stagnant rainwater is admittedly used as drink. Besides, affected localities are found in the province of Posen (diluvial sedimentation with a most impervious substratum), in Silesia, Marienwerder, Königsberg, Merseburg, Talern, flood-and-rivulet parts of the Saale, Elster and Mulde, with a mouldy earth a foot in depth; loose, soft soil, and annual inundations; then in the province of Brandenburg, especially in the neighbourhood of Potsdam (boggy, moorland, humus or sandy subsoil; marshy substrata), in Hessen-Nassau, in the Rhine districts on the alluvium of the Rhine plains (Cleves, Cologne, Bonn); on the Eifel; in the Röhr and Erft valleys (where marshy moors abound); and in the Saar valley.

In Bavaria, the most important anthrax districts are found in the Upper Bavarian Alps, at Miesbach, Tölz, Werdenfels, at a height of from 3,600 to 4,300 feet. These mountain ranges are often deeply fissured and abound in marshy districts, which are for the most part covered with a thick layer of humus, and mingled with disintegrated limestone; the surroundings of the huts is richly furnished with stercoraceous materials. Their spring water is rather warm (50 to 53·6° F.). In addition to the Danube valley, with its huge reeds and mosses, we have further the valleys of the Isar, the Lech, and the Inn.

In Saxony, the district of Zwickau is specially affected, and the disease has recently increased there.

In Würtemberg, anthrax occurs more especially in the Black Forest; Neckar and Jagst districts.

In Baden, the Mannheim locality is notably affected.

In France the noted anthrax districts are: Beauce (Loiret, Eure-et-Loire), the department Seine-et-Marne, Sologne, Burgundy, Poitou, Guyenne, Languedoc, Forez, Lyonnais, Auvergne, Dauphiné, Provence.

In Russia, anthrax is most frequently found in Siberia; also in the governments of Astrachan, Orenburg, Perm, Novgorod, St. Petersburg, Archangel, Finland and Lapland.

Anthrax is also met with in Switzerland, in Austria (Galicia,

Bohemia), Italy, England,* Spain, Turkey, Africa, America, the East Indies, Persia, China, etc.

The money loss from anthrax in cattle is often very great, especially during the so-called anthrax years. From 1872 to 1875 it amounted in Upper Bavaria to £15,000 (over 900 head of cattle and 40 horses). During 1874, in the single district of Potsdam (Prussia), 2,000 red-deer and fallow-deer died from it. The total loss in Prussia from 1878 to 1883 came to more than £75,000 (over 6,000 head of cattle, 3,000 sheep, 300 horses officially registered, not counting numerous cases which had not been reported). In France the annual loss from anthrax amounts to many millions of francs. In former times it was even still greater. Delafond states that in the thirties the annual loss in Beauce alone was 10,000,000 francs (about 5,000 cattle and 300,000 sheep). The department Seine-et-Marne has had since 1859 a yearly loss of about half a million of francs; and the department of Aisne one of 120,000 francs. In the government of Pskow, during 1884, 4,000 horses, nearly 2,000 head of cattle, and 1,000 smaller domestic animals died from it (W. Koch); and in the government of Novgorod, from 1867 to 1868, nearly 40,000 horses, over 8,000 cows, 6,000 sheep, and 500 human beings (Grimm).

Statistics of Epidemics.—In Germany from 1886 to 1898 about 50,000 animals were attacked with an average mortality of from 96 to 97 per cent. Cattle (40,000) suffered most, and after them sheep (7,000). Cases in horses (1,500) and pigs (500) were comparatively rare. There were only 60 cases in goats. The losses in the respective years were as follows:—

Year.	Cattle.	Horses.	Sheep.	Pigs.	Goats.	Total.
1886 . .	2,255	88	542	52	2	2,939
1887 . .	1,977	61	444	30	4	2,516
1888 . .	2,060	40	286	30	3	2,437
1889 . .	2,276	57	622	50	5	3,010
1890 . .	2,537	72	485	26	5	3,125
1891 . .	2,738	69	434	8	8	3,257
1892 . .	3,009	92	561	30	5	3,697
1893 . .	3,010	142	591	27	14	3,784
1894 . .	3,031	373	204	83	8	3,699
1895 . .	3,183	169	551	43	3	3,949
1896 . .	3,709	184	501	26	2	4,422
1897 . .	3,936	148	469	25	0	4,577
1898 . .	4,455	133	293	35	5	4,921
1899 . .	3,678	282	307	61	6	4,334
1900 . .	3,461	142	390	51	6	4,050
1901 . .	4,263	134	1,361	65	20	5,843
1902 . .	4,003	134	620	87	8	4,852

* See foot-note on next page.

Among 10,000 healthy animals we find on an average 1·5 cattle, 0·2 *horses, 0.25 sheep, 0.3 pigs, and 0.02 goats infected.* The increase in the numbers of the recorded cases of the disease which has appeared so prominently in these last years is to be attributed substantially to the introduction of compensation and the notification which has on that account been more frequently made.

Anthrax statistics of foreign states are not so reliable as those of Germany. In Austria, anthrax was particularly rife in Galicia, Lower Austria and Bohemia. In Hungary, during 1889, 2,974 cattle, 8,284 sheep, and 387 horses were attacked; in 1897, 1,889 cattle, etc.; in 1898, 1,888 cattle, 1,110 sheep and 216 horses; in 1899, 2,000 cattle, 200 horses and 600 sheep; in 1900, 2,000 cattle, 200 horses, and 700 sheep.

The general anatomical changes in Anthrax are chiefly as follows:—

1. Hæmorrhages by which all the organs are often permeated.

2. Serous, *gelatinous and hæmorrhagic infiltrations of the* sub-serous (especially of the mesentery and mediastinum), submucous (intestines) and subcutaneous connective tissue.

3. Swelling of the spleen and parenchymatous inflammation of the large glands of the body (liver and kidneys).

4. A tar-like condition of the blood with poikilocytosis and leucocytosis.

5. The presence of the bacillus of anthrax, which is found in all the organs, especially in the capillary blood of the internal organs (spleen, intestines, and liver) and in the serous gelatinous infiltrations of the mesentery.

* The following are statistics of anthrax in Great Britain:—

Years.	Animals Attacked.				Total.
	Cattle.	Sheep.	Pigs.	Horses.	
1893	833	108	313	46	1,300
1894	625	125	188	62	1,000
1895	604	158	140	32	934
1896	632	34	200	38	904
1897	521	39	284	38	882
1898	634	22	161	39	856
1899	634	69	253	30	986
1900	668	40	204	44	956
1901	708	76	152	35	971
1902	746	50	192	44	1,032
1903	809	48	234	51	*1,143
1904	1,115	62	365	47	1,589
1905	1,001	53	210	53	1,317
1906	999	83	213	35	1,330
1907	1,163	66	190	37	1,456

* Including one Deer.

The individual organs show the following changes :—

The vessels of the *skin*, on skinning the animal, appear to be densely filled with dark blood. On closer examination, we may observe under the epidermis and in the tissue of the cutis, small hæmorrhages which in some cases raise the epidermis into the form of vesicles. In anthrax due to inoculation we find in the skin, circumscribed, firm nodules of the size of a lentil up to that of a bean, with partial necrosis of the thickened tissue (the carbuncles of anthrax). Where there is considerable œdema of anthrax, the skin suffers from wide-spread necrosis.

The *subcutaneous connective tissue* is permeated with blood centres the size of a lentil up to that of a shilling, and is of a slightly blood-red tinge. In the œdema of anthrax we find circumscribed or diffuse gelatinous effusions of a rather firm consistence and frequently of a very considerable extent, which vary in colour from orange-yellow to yellowish-brown, or are permeated with spotted and flat extravasations of blood. Also, purely sanious infiltrations occur. The neighbouring lymph glands are considerably enlarged, permeated with hæmorrhages and œdematously infiltrated. The gelatinous infiltrations in the subcutis on the neck and along the trachea down to the chest are very large.

The *muscular apparatus* of the body varies in colour from a dark brownish red to violet, is infiltrated with hæmorrhagic spots, and is very soft. The muscular tissue of the heart shows similar changes (parenchymatous myocarditis).

In the *cavities of the body* (abdomen, thorax, and pericardium) a sanious fluid is found in moderate quantities. Hæmorrhagic extravasations of different sizes are seen under the serous membranes and epicardium, and particularly on the mesentery and mediastinum. The subserous connective tissue, especially on the mesentery, anterior cavity of the mediastinum, and in the neighbourhood of the kidneys, is gelatinously infiltrated, on which account the neighbouring lymph glands are considerably swollen, filled with serum and permeated with hæmorrhages (mesenteric glands and mediastinal glands). The internal organs (spleen, liver, kidneys and lungs) contain a large quantity of blood. All the larger veins and the heart are filled with blood, and the surrounding tissues show sanious saturation.

The *spleen* is considerably enlarged (from 2 to 5 times its normal size), either uniformly or by prominent tumours. The pulp of the spleen is soft, more or less fluid, and stained dark

red. The capsule of the spleen is frequently extravasated with blood, and is always very tense. Sometimes small raised vesicles with sanious contents are to be seen on its surface.

The *liver*, like the *kidneys*, is highly congested and appears somewhat enlarged. The parenchyma is infiltrated with blood-centres and the cells manifest various kinds of degeneration (parenchymatous hepatitis and nephritis). The portal lymph glands often appear enlarged, and the retroperitoneal (perirenal) tissue is infiltrated with a serous, gelatinous fluid. Also, the sub-peritoneal tissue on the intestines and on the abdominal walls is frequently gelatinously or hæmorrhagically infiltrated; and the peritoneum inflamed.

The nature of the lesions of the *intestinal canal* varies according as the disease is intestinal anthrax, or anthrax caused by inoculation. In cases of inoculation-anthrax, the intestine is frequently normal. In other cases there may be subserous hæmorrhages or swelling of the mesenteric glands. The principal changes in intestinal anthrax are always found in the small intestine, chiefly in the duodenum, and more rarely in the colon. In the slighter cases of intestinal anthrax the mucous membrane is affected sometimes by a circumscribed or by a diffuse swelling. It has red spots, is infiltrated with hæmorrhages and covered with erosions, especially on Peyer's patches and the solitary follicles. The bacilli of anthrax are often found in extraordinarily large numbers on the surface of the mucous membrane. Necrosis and ulcers become developed in those parts where the bacilli most thickly congregate. In very severe cases we often find already on the abomasum or on the three first stomachs, gelatinous and sanious infiltrations of the mucous membrane. The mucous membrane of the abomasum, and especially of the duodenum, is, in consequence of excessive hyperæmia, dark red or almost black, and is covered with erosions and ulcers or necrosis, which may extend down to the sub-mucosa. The contents of the intestine are bloody, and the submucosa is infiltrated with a serous, gelatinous, or hæmorrhagic transudate, so that the mucous membrane often projects in the form of large tumours into the lumen of the intestine (œdema of anthrax). On the site of Peyer's patches and solitary follicles, we find flat or prominent nodules (carbuncles), the surface of which is covered with diphtheritic crusts and bacilli. The mesenteric glands are at the same time greatly enlarged and hæmorrhagically and œdematously infiltrated. We seldom find the described lesion in the rectum except when its mucous

membrane has become infected on account of its having been cleaned out with infected hands (carbuncle of the rectum). In this case the mucous membrane is swollen into thick prominences, and is partly necrotic and its surface is covered with blood.

Among the *organs of breathing* the lungs are greatly congested, œdematously infiltrated, and show ecchymoses in some places. The entire respiratory mucous membrane is considerably reddened and affected by ecchymoses. In particular, the mucous membrane of the pharynx and opening of the larynx is often so œdematously or gelatinously infiltrated (angina of anthrax), that excessive stenosis of the larynx takes place. The contents of the trachea and the bronchi consist mostly of bloody froth or mucus.

The *brain* is full of blood and studded with ecchymoses, and the surface of its membranes often exhibit hæmorrhages with an accumulation of sanious serum in the ventricles. Extravasations of blood sometimes occur in the anterior chamber of the eye and under the retina. All the other organs (the sexual organs, urinary organs, salivary glands, thyroid glands, bones, articulations, etc.) show hæmorrhages, and the urine frequently contains blood.

The *blood* is dark-red or almost black, has a tarry or varnish-like lustre, is of a watery consistence, and shows very little tendency to coagulate. It does not assume its normal red colour when exposed to the air. The red blood corpuscles appear in various stages of disintegration, and consequently their forms are very irregular (*poikilocytosis*). The number of the leucocytes is considerably increased (*leucocytosis*). Between the blood corpuscles we find, especially in the blood of the spleen, a considerable number of anthrax bacilli. The bodies of animals which have died from anthrax are often well nourished; they remain limp for a long time; grow cold very slowly (absence of rigor mortis); become quickly decomposed; and are, as a rule, greatly distended with gas. The visible mucous membranes are of a purple colour; blood flows from the natural openings of the body (mouth, nose and anus); and the rectum is sometimes prolapsed.

All the foregoing lesions may be absent in very acute cases (apoplectic form of anthrax). The characteristic bacilli of anthrax are, however, always present in the cadaver.

General Symptoms of Anthrax.—The symptoms of anthrax vary not only in the different species of animals, but also in individual cases, according as the intestinal canal, skin, lungs, etc., are respectively infected. It is even possible that the symptoms of intestinal anthrax may be quite different from the usual ones of this variety of the disease ; or symptoms of metastatic affections of the intestine or serous membranes may become prominent in anthrax of the skin. The nature of the affection may vary essentially according to the amount of the virus of anthrax received by the animal body. Abortive cases and remittent forms of anthrax may also occur.

The characteristic features of all these forms of anthrax are the suddenness of the attack ; severity of its course, which usually ends in death in from 1 to 3 days ; grave general disturbance ; high fever ; tendency to hæmorrhage of the mucous membranes ; and the presence of the bacilli in the blood. To these we must add the different local symptoms, such as carbuncles and œdema of the skin ; intestinal trouble ; brain complications ; and difficulty in breathing. Anthrax has been classified according to its course (*anthrax acutissimus, acutus*, and *subacutus*), or according to the presence or absence of local manifestations. We consider the latter classification to be more appropriate than the former for clinical purposes.

Forms of Anthrax without Visible Localisation.—These forms, which can be divided into peracute, acute, and subacute, are generally due to ordinary infection by spores or to experimental feeding with spores.

1. The *peracute form* is known as *apoplectic anthrax*, and gives rise to symptoms of cerebral apoplexy. The animal becomes suddenly ill, staggers, falls down, often gives vent to a bloody discharge from mouth, nose, and anus, and dies in convulsions after a period of illness varying from a few minutes to 1 hour at the most. Frequently the patient is found dead in its stall in the morning, or may die suddenly while at work, pasture, or feeding. This form occurs most frequently in sheep and cattle, and particularly at the commencement of an epizoötic.

2. The *acute form* runs a somewhat slower course and lasts from 2 to 12 hours, the maximum being 24 hours. The animal soon becomes feverish (104° to 107° F.) with an extremely rapid rise of temperature, and shows either signs of congestion of the brain or of congestion of the lungs. In the

former case, the chief symptoms are: restlessness, excitement, stamping, kicking, rearing, bellowing, attacks of fury, running to and fro, convulsions, spasms, grinding of the teeth, followed by stupor, insensibility, staggering, and finally death as if from apoplexy. In the latter case, the usual symptoms are greatly accelerated and difficult breathing, wheezing, panting, groaning, palpitations of the heart, excessively frequent and very small or imperceptible pulse, cyanosis of the mucous membranes of the head, bloody discharges from the natural openings of the body, hæmaturia, staggering, convulsions, and finally death from suffocation. Sometimes there is a partial remission of the symptoms with a relapse after a short time (*anthrax remittens*). In some cases we observe, before the commencement of the stormy course of the disease, premonitory symptoms of diminished vivacity, slight disturbances in digestion, constipation, tenesmus, etc.

3. The *subacute form* is termed *anthrax fever* or intermittent anthrax, and is the ordinary kind which attacks horses and cattle. The symptoms are generally the same as those of the acute form, except that they are more clearly defined and the course is longer; 24 to 48 hours being the average duration, and 5 to 7 days the maximum. The phenomena of fever (rigors, unequally distributed temperature, and general disturbance of health) and those of congestion of the lungs and brain are more clearly marked, and are frequently complicated with severe intestinal troubles, especially colic. They may abate so frequently that the disease may be erroneously supposed to be of an intermittent nature.

Forms of anthrax with visible localisation appear to arise usually from infection by bacilli, as they can be experimentally produced in the form of carbuncles by inoculation.

The *carbuncles and œdema of the skin*, which are known as "*carbuncle disease*," chiefly occur in horses and cattle, and may also appear in dogs. The carbuncles are circumscribed cutaneous swellings, which are at first hard, hot, and painful; and later on, cold, painless, and tending to become gangrenous. The œdema of anthrax consists of diffuse, flat, doughy, cold, painless, and frequently fluctuating swellings of the skin. The duration of the disease is from 3 to 7 days. Recoveries are more frequent than in cases of anthrax without external manifestations. Fever may appear before or after the formation of the swellings.

Carbuncles and œdema of the mucous membranes are mostly found on the mucous membrane of the mouth (anthrax of the tongue), in the pharynx and larynx (*anthrax angina*), and in the rectum. At the same time the patient suffers from fever, dyspnœa, laryngeal stenosis, difficulty in swallowing, general cyanosis, swellings in the intermaxillary space, and on the throat and the chest, painful straining to defecate, etc. Death takes place in from 12 to 24 hours. This form of anthrax is most frequent in pigs and dogs.

Incubation Stage.—There have been no conclusive researches as to the duration of the incubation of anthrax, in the usual form of infection of horses and cattle from the intestine. Recent feeding experiments carried out among sheep with spore-bearing bacilli, have shown that with rich spore contents the sheep, without exception, die within 2 days after the feeding. In former experiments of feeding sheep with anthrax fodder, this interval lasted from 2 to 3 days. Accordingly, the incubation stage proper among sheep is about 2 days. With regard to the peracute course of anthrax in sheep, the interval which occurs between the infection and the onset of the first symptoms of the disease (that is to say, the incubation stage proper), is almost identical with the period of time between infection and death. Clinical observations (various pastures in an infected locality) and some experiments on feeding with spore-containing material, have shown that the incubation period in cattle and horses also appears to last for from 2 to 3 days.

The usual forms in cattle are: "apoplectic anthrax" (Vol. II., p. 540), "anthrax fever" (p. 541) and "carbuncle disease" (p. 541); in horses, the same forms, and especially anthrax fever; in sheep, apoplectic anthrax; in dogs, carbuncles; and in pigs, anthrax angina and anthrax of the tongue. We should be cautious in accepting supposed cases of anthrax in pigs.

General Diagnosis.—In the acute or subacute form, a correct diagnosis during life can be made only by a microscopical search for the bacilli, or by the knowledge of the epizoötic character of the disease. A bacteriological examination of the blood very frequently gives a negative result, because the bacilli chiefly congregate in the internal organs. It has consequently been proposed to examine the blood of the liver after puncturing that organ with a trocar and cannula. The difficulty of the diagnosis *intra vitam* is further increased by the fact that the symptoms of the disease somewhat resemble those of simple congestion of the lungs or brain (although anthrax is to be distinguished from them by the presence of high fever), poisoning, rapidly appearing septicæmia, etc. Consequently an exact diagnosis can, as a rule, be arrived at only after death. The demonstration of the bacilli is generally necessary for the

anatomical diagnosis; for the microscopical result is rarely sufficient by itself. For the demonstration of the anthrax bacillus, we may best employ one of the following methods.

1. Johne recommends the following method for the detection of the bacilli: the cover glass preparations, which have been prepared in the usual way, and have been drawn three times through the flame, are stained for about a quarter of a minute (at the utmost for half a minute) by dropping a 2 per cent. watery solution of gentian violet on the cover glass, which must be cautiously warmed, along with the solution, washed for a moment in pure water, and afterwards for 8 or 10 seconds in a 2 per cent. watery solution of acetic acid, and finally very carefully washed again in pure water. A drop of water is then applied and the preparation examined with a magnifying power of at least 400 times.

2. Klett advises that the cover glass with the blood spread out on it should be allowed to dry in the air for some hours, drawn 3 times through the flame, stained for a few seconds with a watery solution of gentian and rinsed in water. After this, distilled water is dripped on the stained side of the cover glass, which is once more warmed by being passed from 6 to 12 times through the flame. It is rinsed again, placed on the object bearer, and examined when the upper side has become dry. By both methods the characteristic capsule of the bacillus of anthrax is made distinctly visible.

3. In Olt's method the spread-out preparation is dried in the air, passed 3 times through the flame, and then stained with a 3 per cent. watery solution of safranin; after which it is within half to one minute held three times over the flame till bubbling of the staining solution takes place. The anthrax bacilli then appear as reddish-brown bacteria cells, with brightly glistening corpuscles and a quince-yellow gelatinous capsule. Olt's method is as simple as it is reliable.

4. In Raebiger's method the spread-out preparation is not heated, but in the air-dried condition is moistened with some drops of a formalin-staining solution (15 grams of gentian violet with 100 grams of formaldehyde) (= 4 drs.: 3½ oz.), which is left on the preparation for twenty seconds, and then washed off with water. When examined with an immersion lens, the bacteria and cells appear densely stained, more deeply and strongly than in the other staining methods. The capsule especially is very broad and easily recognisable from its blue contour. Caution should be used by the operator on account of

the strongly irritating properties of formaldehyde on the skin and mucous membrane.

Inoculation is another aid to diagnosis, the most suitable animals being mice, rabbits and guinea-pigs. Sheep, if available, are good subjects for inoculation. The diagnosis is confirmed by the occurrence of death after 2 or 3 days, and by the not very difficult demonstration of the bacilli in the cadaver. We may diagnose intestinal anthrax by inoculation with the fæces of the infected animals. An ordinary small wound in the skin will suffice for inoculation with the suspected material (blood, mucus, fæces, etc.). In some cases, inoculation with material from affected animals is unsuccessful, as for instance with blood from the surface of the body or larger blood-vessels which contain no bacilli; when the inoculation material is mixed with other bacteria; or when it is in a state of decomposition. All kinds of animals are not equally suitable for inoculation. Even in the same species of animals there may exist great differences of comparative immunity owing to breed or individual idiosyncrasy. Thus, very old animals are much less susceptible than very young ones. The technique of the inoculation, notwithstanding its great simplicity, may be defective, as when the introduced virus has been removed by the outflowing blood, etc. Several young animals and, if possible, different species of animals should be inoculated at the same time.

Uses of Blood-testing in the Diagnosis of Anthrax.—As a rule, the bacteriological recognition of the bacilli suffices for the definite establishment of the existence of anthrax. A complete dissection of the cadaver is accordingly unnecessary, when by testing some blood from the jugular or aural vein, or taken from the spleen by means of a grooved trocar, the presence of anthrax bacilli in the blood can be established beyond dispute. A complete *post-mortem* is to be avoided as far as possible, as the dissemination of the anthrax germs is promoted by opening the body. Only when the bacteriological examination of the blood taken for the purpose has proved negative or doubtful, should a complete dissection of the body be undertaken in order to decide the cause of death.

Slaughtered animals present, in the first place, changes in certain organs which give reason to suspect anthrax; especially the spleen (swelling); also, the epicardium (hæmorrhages), and the intestinal hæmorrhagic inflammation. In such cases an examination of the bacilli from the spleen with a simple bacteria microscope suffices to establish a positive diagnosis. Such microscopes are in the possession of nearly all veterinary practitioners. With a little trouble, spread-out preparations may be made for control preparations.

If only single pieces of flesh are at our disposal, specimens from the deep parts of the same, preferably traces of blood from a vein situated

in its interior, are examined for the presence of anthrax bacilli. In order to avoid confusion of the anthrax bacilli with other bacteria, inoculation must be carried out in doubtful cases, with subsequent bacteriological examination (rabbits, white mice), and production of pure cultures. The inoculations and pure cultures are always necessary for the establishment of diagnosis when the carcases of animals, which have died with symptoms suspicious of anthrax, have not been cut open.

The packing of anthrax material for the purpose of transport or of special examination, is best carried out in the form of thin and thick spread-out preparations and slides. The thin spread-out preparations, dried in a few minutes by exposure to the air, or rapidly over a flame, serve for staining; while the thick ones can be used for the preparation of cultures, and as material for inoculation experiments (Bongert, Hosang). The transmission of anthrax in corked flasks, which was formerly usual, is not to be recommended, on account of the early occurrence of decomposition (shrinkage and disintegration of the anthrax bacilli, negative results of cultures and inoculations). The period of time within which the anthrax bacilli perish through decomposition is also not always the same. In unopened cadavers they perish more rapidly (anærobic decomposition) than they do in bodies that are opened early.

Plate Method of Bongert.—According to the researches of Bongert, the recognition of the anthrax bacilli in spread-out preparations, is by itself, in many cases, no sure evidence of a positive diagnosis. Diagnostic inoculation, also, on account of the antagonistic influence of secondary bacteria, often fails us. The surest and best method of bacteriological diagnosis of anthrax is the plate procedure. His experience is that anthrax bacilli remain active for from 36 to 50 days in dried blood, and for from 8 to 20 days in decomposing blood. The best way of preserving anthrax material for future bacteriological investigation, is carried out by prolonged drying in thick layers on slides, or on the inner surface of test-tubes. Under these conditions, the anthrax bacillus preserves its power for at least from 10 to 14 days, and its capability for propagation. The demonstration of the bacilli in spread-out preparations can, in case of cadavers which have remained unopened, become uncertain and inconclusive even within 24 hours after death. On the other hand, by the plate method, the bacilli can, in such cases, be easily and surely recognised even in from 1 to 3 days after death.

Malkmus found that microscopic investigation and animal inoculation very soon fail to give results in case of decomposing material; in such cases those of dissection and the course of the symptoms of the disease are more conclusive. On the other hand, the plate procedure of Bongert usually gives good results in decomposing material; it must not, however, be regarded as the only reliable one. Berndt and Fischöder state that the staining method of Klett yields the best evidence in stale material; while the other staining methods (Johne, Olt, Raebiger, and others) fail at an early stage. Fischöder found the demonstration of the bacilli in cadavers often difficult, as they rapidly diminish in number and undergo changes. No method of bacterial demonstration should therefore be neglected. The diagnosis of anthrax in practice is by no means simple and easy. A *post-mortem* examination should, Fischöder holds, be carried

out in every case. With regard to compensation, the objective condition found on dissection must form the sole basis of legal right thereto.

Verification of the Diagnosis of Anthrax.—Ostertag found that the diagnosis of the disease in animals condemned to slaughter for anthrax, and which have been disembowelled directly after slaughter, can be established with certainty by the microscopic examination of stained spread-out preparations made from the fresh or dried spleen-pulp. The anthrax bacilli are in these cases present in such abundance, and can by special methods (Johne, Klett, Olt, Raebiger, and others) be stained so characteristically, that no doubt of the presence of anthrax can exist. Similar results may be often obtained with blood taken from the veins of the neck or head of animals which have died of anthrax, when the materials for investigation were procured immediately after death. In all these cases, a further investigation of the transmitted material for confirmation of the diagnosis is not necessary. The further research is not, however, to be abandoned when the microscopic testing leaves the slightest doubt as to the presence of anthrax. Such doubt can be removed by inoculation and the arrangement of plate cultures.

The anthrax bacilli produce no spores either in the animal body or, as a rule, in the matter withdrawn therefrom. They are influenced to such a degree by the bacteria which appear in the organs and blood vessels, as the result of cadaveric decomposition, that the number of the anthrax bacilli recognisable by the aid of the microscope continually diminishes. The material then no longer kills mice by anthrax; and, finally, anthrax colonies cease to develop even in prepared cultures. For instance, in sheep which have died of anthrax, while great numbers of the bacilli can be recognised immediately after death, they are found to have so completely perished before the end of 3 days, that a positive recognition cannot be established, either by microscopic investigation, or by inoculation, or by culture, when the bodies have remained unopened in presence of an elevated external temperature. As the cultures made from materials which have ceased to kill mice by anthrax still continue to give positive results, cultures should always be prepared in cases of anthrax, in which the microscopic examination has left the decision doubtful. The colonies which develop from the still active anthrax germs contained in the materials, are of a form so characteristic, that by a mere glance at the cultures with a slightly magnifying power the diagnosis is assured. In doubtful cases, pure cultures can be prepared from the developed colonies, and may be further studied. For plate cultures, comparatively large quantities of the separated materials can be used, as the cadaveric bacilli do not develop on the plates; and by the presence of such putrefactive bacteria as develop on the plates, an extended assortment of the germs may be carried out by the employment of diluted cultures. For the inoculation of mice, it is advisable to use but a small quantity of material, as powerful decomposing matter may kill mice, if given in large portions. Both for the preparation of plate cultures and the inoculation of mice, it is recommended to use the transmitted blood-test; because the anthrax bacilli contained in the veins of the head or neck are less affected by cadaveric bacilli, in the unopened state of the body, than are those in the spleen-pulp.

As already indicated, the anthrax bacilli may, even in a relatively short space of time, be so completely destroyed by the natural processes of disintegration within the unopened body, that a positive demonstration cannot be attained by any of the methods known to bacteriology. When the cadaver has remained unopened for from 1 to 2 days before the removal of the material, or when the removal, in the case of disembowelled animals, has taken place so late that an advanced degree of disintegration of the materials of investigation has already taken place, the refusal of compensation, on the ground of a negative or a doubtful result of the bacteriological examination, cannot be justified. In such cases, the decision has to be made on the results of the dissection, and circumstances under which the death occurred. Therefore it is advisable in cases of negative or doubtful results of bacteriological examination, to form a decision on the grounds of the data furnished by the symptoms of the disease and the evidences of dissections.

Differential Diagnosis in General.—As the various diseases which may be mistaken for anthrax will be discussed in detail when referring to the respective species of animals, we shall here content ourselves by giving the following brief description of those bacilli which possess some similarity to the bacilli of anthrax.

1. The *septic bacilli*, or the *bacilli of decomposition* (especially *bacterium termo*) are distinguished from the bacilli of anthrax, usually by their being motile, and by their rounded ends. Other bacilli which closely resemble the bacilli of anthrax, and can be distinguished from them only with difficulty, are sometimes met with. In such cases, the peculiar morphological relations, especially the presence of the capsule of the bacilli of anthrax, must be looked for, or test inoculations must be made.

2. *The bacillus of quarter-ill* is shorter and stouter than that of anthrax ; the ends are rounded ; and the bacillus is very motile, forms gas, and produces in cultivations variously folded dirty-white membranes. Being an anærobic fungus it cannot be cultivated on potatoes.

3. *The bacillus of malignant œdema* is thinner than that of anthrax ; it is not articulated, and has rounded, not cut-off ends ; it does not occur in the blood ; it usually possesses slowly rocking movements ; and grows out into very long and, frequently, circularly arranged threads, which are free from spores, and produces bubbles of gas in the cultures. A piece of tissue containing the bacilli of œdema placed in gelatine, forms around itself a white globe of fluid, the surface of which seems to be covered with fine needles. This bacillus can be cultivated only with great difficulty in laboratories (Kitt).

4. *The bacillus subtilis* (hay bacillus) is thick and short, shows oscillatory movements, and has a stout flagellum at both ends. This bacillus develops from a spore perpendicular to its longitudinal axis. Although it is ærobic, it is of much less importance in the differential diagnosis of anthrax than the other fungi we have mentioned (Kitt).

Prognosis in General is very unfavourable; the mortality being on an average from 70 to 90 per cent., and 100 per cent. in apoplectiform anthrax. After the epizoötic has continued for some time, the disease sometimes assumes a milder type. Some animals acquire immunity for a short time from a previous attack. Recoveries without any treatment are not rare.

Therapeutics in General.—Prophylaxis, which is much more important than treatment in cases of anthrax, consists first of all in the thorough destruction of the anthrax cadaver, and in the careful disinfection of the contaminated stalls. The destruction of the cadaver is best carried out by burning, when possible, or by burying it at least at a depth of 6 feet or 7 feet, and at some distance from roads, houses, and pastures. It may also be rendered harmless by steaming under pressure sufficient to break up the soft parts.

An attempt to reduce the infectiousness of anthrax stricken districts should be made before all things by a rational improvement of the soil. In this view, the draining of swampy and damp ground, regulating the water supply, and the closure of all suspected pastures and suspected wells, must be considered as being of the first importance. A change of habitation can seldom be carried out as easily as one of food and water. The medical treatment, which is not always successful, consists in the internal application of hydrochloric acid, creolin, carbolic acid, or salicylic acid. Medicines are not of very much use; although almost every agent in the pharmacopœia has been tried. Meier has reported strikingly favourable results from creolin in 5 cases of anthrax in cattle; and in 5 other cases, instantaneous improvement and quick recovery from the administration of 50 to 200 grammes (1¾–7 oz.) of creolin. This curative action of creolin in anthrax has been confirmed by Hansen, Schneider, Bucher, Struwe, Witt, Seiffert, Wancke, Wolf and Persenaire. Also in the publications on the subject by the official veterinarians of Prussia in 1900. The writers report

most favourably on the internal administration of creolin in anthrax. The reports for 1901 testify also to numerous cases of cure of anthrax by creolin at Flatow, Marienberg, Schubin, Striegau, Namslau, Haynau, Sonderburg, Warendorf, Essen, Mörs and Euskirchen. The following agents have also been recommended: corrosive sublimate (subcutaneous injection); carbolic acid and salicylic acid (10 to 15 grammes (154–231 grains), or more *per os* in cattle); iodine in the form of Lugol's solution, namely: iodine 30 grammes (7½ drms.), iodide of potassium 60 grammes (1 oz. 7 drs.), distilled water 360 grammes (12 oz. 6 drs.), of which 2 tablespoonfuls in a quart of water is to be given every 2 hours: argentum colloidale (intravenous); chlorine water; calcium chloride; arsenic; phosphorus; hydrochloric acid; liquor ammoniæ; oil of turpentine; sal volatile, etc. It is customary in the subcutaneous form of the disease to administer neutral salts and other aperients, and small doses of calomel. In congestion of the lungs and brain, phlebotomy deserves a trial.

The carbuncles of anthrax on the skin can be treated by long and deep incisions with subsequent application of disinfecting remedies, or the actual cautery.

Pasteur's Protective Inoculation against Anthrax.—It had previously been known that many animals, after recovery from one attack of anthrax, continued to enjoy an assured immunity from subsequent attacks of the disease. Certain animals appear to possess a natural immunity from anthrax, as is the case with Algerian and Berber sheep; specimens of individual immunity are also found. On the other hand, there is no way of immunising guinea-pigs, rabbits, rats or mice (Löffler); and horses can be rendered immune only with great difficulty, as the researches of Oemler have clearly demonstrated. He produced anthrax by successive inoculations 7 times in the same horse. Man also possesses no immunity. It is, however, possible to produce a certain immunity in sheep and cattle by inoculation; even though it be but partial, and of relatively short duration.

The first protective inoculations were carried out by Toussaint. He warmed defibrinated anthrax blood for from 10 to 15 minutes in a temperature of from 122° to 131° F. and then used it immediately for inoculation; he had, however, no definite idea as to the cause of its immunising effect. Pasteur first pointed out that immunitiy was due to a weakening (mitiga-

tion) of the bacilli. The virulence of the anthrax bacilli may be weakened by various reagents ; by influence of heat (Pasteur, Toussaint, Chauveau) ; compressed oxygen (Chauveau and Wosnessenski) ; by antiseptics (Chamberland and Roux) ; by sunlight (Arloing). It can also be weakened by culture of the bacilli on the blood of inoculated wethers (Metschnikoff), or the bodies of frogs (Lubarsch). Pasteur prepares his inoculation material (vaccine) by culture of the bacilli at from 107.6° to 109.4° F., in oxygen. Then he uses a first, weaker ("primary") vaccine, which is prepared by a culture of 24 days, and a second, stronger ("secondary") vaccine, prepared by a culture lasting for 12 days at the same temperature. The animals are first inoculated with the primary vaccine ; and then, after an interval of from 10 to 14 days, with the secondary vaccine.

The results of the protective inoculations which have been carried out according to Pasteur's method in all the countries of Europe (France, Russia, Hungary, Germany, Italy, Holland, Belgium) and also in America—including many hundreds of thousands of cattle, have proved to vary to an extraordinary degree. They are briefly as follows :

1. Pasteur's protective inoculation is not, as a general rule, to be recommended for sheep, as it frequently produces either no immunity, or one of very short duration. In the most successful cases this immunity would last but a year, so that annual inoculation would be requisite. The mortality of successful inoculation, also, is too great, and in some cases reaches in the second inoculation a proportion of from 10 to 15 per cent. The results of Pasteur's inoculation are very unequal ; as in one case it is too strong, and in another too weak, according as the temperature ranged closer to 107.6° or 109.4° F. Koch advises, therefore, that the Pasteur vaccines should always be tested, before use, in this way : the primary vaccine should kill mice, but not guinea-pigs ; while the secondary vaccine should be fatal to mice and guinea-pigs, but not to rabbits. Sometimes the weakened ("mitigated") inoculation matter becomes again virulent, especially when the weakening has been rapidly induced by a high temperature ; while, on the other hand, it may completely lose its power by lengthened delay in its use. A special preparation of inoculation matter is, therefore, necessary every time for sheep ; while, again, we have the fact that some breeds of sheep are unequally susceptible to the same vaccine. Finally, the expense of inoculation has to be also considered. From the favourable results obtained in

France and Hungary, protective inoculation of sheep—with reservation and the condition of using healthy inoculation material—is to be recommended experimentally, only when we have to deal with a permanently infected locality, with regular and considerable losses (at any rate, over 2 per cent.).

2. The Pasteur inoculation is not associated with such great losses in cattle as in sheep. Its employment on cattle in the experiments carried out in Prussia caused considerable losses, on account of the varying effects of the individual vaccines, which are specially great when the first vaccine has been made too weak at 109.4° F., and the second too strong at about 107.6° F. We must also consider the uncertainty and the relatively short duration of the results of the inoculation (one year only, and sometimes even less); the danger of the spreading of the anthrax bacilli through the extremely virulent bacilli of the second vaccine, in non-inoculated animals or in man, and also the commercial losses due to the illness of inoculated animals. Horses, especially, bear protective inoculation very badly. Whether the serum-inoculation (Sobernheim) will yield better results remains still to be seen (see Vol. II., p. 354).

Technique of the Inoculation.—The technique of Pasteur's method is as follows: The tubes coming from Pasteur's laboratory in Stuttgart or from Boutroux, 28 rue Vaucquelin, Paris, and costing £1 for fifty cattle or one hundred sheep, must be used at once and at one sitting, and must be opened only immediately before the inoculation, for which we use a Gram syringe. After the syringe has been well cleaned and disinfected before each application, the lymph tube which contains the *premier vaccin* is well shaken, the plug taken out, and the syringe filled directly from the tube. One eighth of the contents is subcutaneously injected on the inner side of the right thigh of the sheep, and the puncture is closed with the thumb. After 12 to 14 days the second inoculation is performed in the same way with the *second vaccin*. In cattle, a quarter of the contents of the syringe is injected behind the right shoulder with the *premier vaccin* and behind the left shoulder with the *second vaccin*. The hair should be removed from the site of the inoculation before the injection is made. Lambs, calves, and cows advanced in pregnancy should not be inoculated.

Chauveau has lately introduced a method for the rapid preparation of Pasteur's vaccine by heating the cultivations for 10 hours at 109° F.; for 3 hours at from 116° to 120° F., after renewal of the nutrient material; for 2 days at from 95° to 98° F. (for the formation of spores); and finally he raises the temperature to 176° F. He has also proposed attenuation by compressed air. Chamberland and Roux weaken the bacilli by chemical agents, as for instance, by the action of carbolic acid (1 to 600) for 24 days, or of bichromate of potash (1 to 2,000–5,000) for 10 days. Gibier makes his inoculation material by reducing the temperature of the bacilli to − 40° F. Kitt attenuated the bacilli by passing

them through the bodies of pigeons. Chauveau and Perroncito have made vaccines respectively for cattle and sheep by only one (instead of two as hitherto done) heating of the bacilli at from 98° to 100° F. for 5 days. According to Chauveau's latest method, the point of the glass tube containing the vaccine is broken off, and one drop for each animal is injected with a Pravaz syringe on the upper surface of the cartilage of the ear. Arloing, Cornevin and Thomas consider that the attenuated virus of anthrax may be restored to its former strength by the addition of lactic acid. Nocard and Roux believe that this increase of virulency is not due to any influence on the bacilli, but to a reduction of the power of resistance of the tissue cells by the action of the lactic acid.

Statistics of Inoculation.—The following statistics relate to the most important protective inoculation experiments which have been carried out during the last few years :—

1. In *France*, which is the fatherland of anthrax inoculation, during the years 1882–1893 not less than 3,296,815 sheep and 438,824 cattle were inoculated. Out of that number, reports were received concerning the results of 1¾ million cases of sheep, and 200,000 cases of cattle. The mortality in cattle amounted to 0·34 per cent. ; and in sheep to 0·94 per cent. Before the introduction of inoculation, 5 per cent. of all the cattle and 10 per cent. of all the sheep were said to have died from anthrax. The financial gain from inoculation is estimated in cattle at £80,000, and in sheep at £200,000.

2. In *Austria* 350,000 sheep and 130,000 cattle were inoculated *à la* Pasteur during the years 1882–1887, with a decrease in mortality, it is stated, of from 10 to 14 per cent. to from 1 to 2 per cent. In Hungary, in the years 1889–1895, 500,000 sheep (loss due to inoculation=1·1 per cent.), 160,000 cattle (loss=0·1 per cent.), and 10,000 horses (loss=0·26 per cent.), were inoculated with, professedly, very good results. In the years 1899–1900, the direct losses produced by inoculation (including both operations) were : in sheep, 0·26 per cent. ; in horses, 0·1 per cent ; and in cattle, 0·02 per cent.

3. In *Russia* 20,000 sheep were inoculated in the district of Cherson in 1888 (Skadowski), with a loss which was due to the inoculation, of 0·5 per cent. of the inoculated sheep, and 0·3 per cent. subsequently died from ordinary anthrax ; making a total loss of 0·8 per cent. It is stated that the loss from anthrax among non-inoculated sheep amounted to from 10 to 33 per cent. ; and that ewes inoculated after the third month of pregnancy produced immune lambs. The loss due to the inoculation in cattle (155 head) was 0·75 per cent. Skadowski recommends the adoption of protective inoculation in all places where the annual loss from natural anthrax amounts to 2 per cent. or more. In the Crimea, 4,564 sheep were inoculated in 1888 with vaccine obtained from Odessa (Bardach), the result being that 3,478 (80 per cent.) sheep died owing to a mistake having been made between the *premier* and *second vaccin*. Inoculations made with Pasteur's vaccine produced similar unfavourable results in the district of Odessa (Gamaleïa). Wysokowicz states that in the district of Cherson the loss due to inoculation *à la* Pasteur amounted at first to nearly 2 per cent. in 1885 ; but from that year to 1888, out of 20,000 sheep which had been inoculated, only 170 (0·87 per cent.) died. According to Woronzow 1·25 per cent. of 7,000

inoculated sheep died in 1890 ; but none out of 200 head of cattle. It is stated that the previous annual loss was from 10 to 12 per cent. Podmolinoff reports that during 1892 and 1893, 67,000 sheep were inoculated in the district of Cherson with a loss of 0·43 per cent. ; 1,450 horses with a loss of 8 animals ; and 3,652 cattle, with a loss of 2. According to Ekkert, 38,936 sheep were inoculated in Southern Russia from 1883 to 1889 with a loss of 0·53 per cent. In the year 1890 the loss varied in inoculated adult sheep from 0·03 to 0·3 per cent. ; in lambs, from 0·1 to 3·6 per cent. ; and in particularly well-bred lambs the loss reached 13·3 per cent. ! The losses through natural anthrax fell in the inoculated flocks from 8–12 per cent. to about 0·1 per cent. Lange inoculated 928 animals without any dying ; and Gordsälkowski, 24,500 sheep with a loss of 0·3 per cent. Beresow states that in 1890–1891, 68,287 sheep were inoculated with a loss of from 0·3 to 0·8 per cent. ; 916 horses, with a loss of 0·3 per cent. ; and 1,449 cattle without any casualty. Of the inoculated sheep, 2·2 per cent. died from natural anthrax after the inoculation. In 1897, the best results were obtained with the spore-vaccines of Cienkowski (Charkower vaccine). In 1900, 115,000 cattle were immunised with Lange's (Kasan) vaccines (including 40,000 cattle and horses, 30,000 sheep, 1,000 camels, etc.). Ekkert made a report on the inoculation of reindeer (protective inoculation, not forced inoculation).

4. During the years 1882–1888 in *Prussia*, (Emler inoculated *à la* Pasteur 841 cattle and 3,459 sheep with a mortality in the former of 0·3 per cent. ; and in the latter of 0·9 per cent. Subsequently 2·8 per cent. of the inoculated cattle and 1·9 per cent. of the inoculated sheep died of anthrax ; consequently the total loss of the inoculated cattle was 3·1 per cent., and of the inoculated sheep, 2·8 per cent. Barkow inoculated, in 1898, 330 cattle with Pasteur's lymph in West Prussia (Stuttgart Depôt). There was a loss of only one from the effects of the inoculation. After the first inoculation with both vaccines, some occasional losses did, however, occur ; but they ceased on the repetition of the inoculation. On that account Barkow recommended a double inoculation,

5. In *Italy*, that former zealous advocate of inoculation, Vicentini, now speaks unfavourably of it. In the province of Belluno, 18 out of 168 inoculated animals died. Other inoculations in 3,000 cattle caused decrease of milk, grave œdemata in a few cases, and some abortions and deaths. On the other hand, in the years 1897–1899, 90,000 cattle and 220,000 sheep were inoculated in 85 provinces with good results. A more recent (1900) vaccine of treble strength, prepared by Melloni, is reported to have given good results.

6. In *Würtemberg* 40 cattle were inoculated without any loss in 1891. In 1900, 184 head of cattle were inoculated.

7. In *Holland* 255 cattle were inoculated during 1889–1890 with one death from anthrax. In 1893–1899, 1,600 cattle were inoculated in North Holland with results which were reported to have been most favourable.

8. The inoculation experiments which McFadyean made in *England* have convinced him that it would be inadvisable to introduce Pasteur's method into that country.

9. In America, since 1896, inoculation has been practised with good results (in 1900, 50,000 doses of lymph were employed).

Serum Inoculation.—According to the investigations of Ogata

and Jasuhara, the protective and curative effect which has been obtained in certain infective diseases (tetanus, erysipelas, and human diphtheria) from blood serum taken from patients that had suffered respectively from these diseases, may also be obtained in anthrax. These observers state that even the blood of animals which are immune to anthrax (dogs, white rats, and frogs) is able to weaken the virulence of the bacilli of anthrax outside the animal body. Ogata extracted from the blood of dogs by means of alcohol and ether a ferment-like substance which had disinfecting properties, and which rendered animals immune to anthrax. As Enderlen, Kitt, and Grabitschewsky were not able to confirm these observations, we must wait for further results in serum-therapeutics. According to the more recent researches of Sobernheim, a sure protection of sheep and cattle against infection with highly virulent anthrax bacilli may be obtained by the serum of artificially immunised animals. The advantages of Sobernheim's method over that of Pasteur are: its safety, simplicity, more prolonged influence after inoculation, and also of curative power. Both the immunisation by serum alone and the combined employment of serum and fully virulent cultures have proved to be reliable in sheep and cattle. In the province of Pomerania, 2,700 cattle were inoculated by Sobernheim in 1902, without any loss and with conspicuously favourable results. Burow published results of 5,000 cases of inoculation carried out by him in 1903, according to the Sobernheim method: the serum was first injected; and 5 minutes later a culture (simultaneous inoculation); 8 oxen (= 0·15 per cent.) died of the inoculation anthrax, 3 cows (= 0·08 per cent.) died from 4 to 6 weeks after the inoculation. Kunze has inoculated 240 cattle and 28 horses with professedly good results. Another serum is recommended by Mendez; 0·5 to 1 c. cm. (8½ to 17 mins.) of it will effect a cure in sheep and cattle. Emmerich, on the strength of his experiments with rabbits, recommends at an outbreak of anthrax, prophylactic inoculation of cattle, sheep, and other animals, with attenuated cultures of erysipelas. He states that the cocci of erysipelas increase the activity of the cells in an anthrax-infected organism to such a degree, that the tissues can successfully combat the bacilli of anthrax. Pawlowsky says that the coccus of pneumonia, the *staphylococcus aureus* and the *bacillus prodigiosus* have a similar effect.

B.—ANTHRAX IN THE SEVERAL DOMESTIC ANIMALS.

(1.) *Anthrax in Cattle.*

Symptoms.—Anthrax in cattle usually assumes the acute form without any outward manifestation. There is a sudden accession of high fever, with an internal temperature of 105·8° or 107·6° F.; the pulse is very frequent (80 to 100, or even more, in the minute), small, and scarcely perceptible; the visible mucous membranes of the head are greatly congested, and are frequently cyanotic; the conjunctiva is often infiltrated with hæmorrhages and considerably swollen; and the eyes are filled with tears. The external temperature of the body is unequally distributed;

the hair stands on end ; feeding and rumination are suspended : and there is great depression, weakness, stupor, and loss of sensation. The animal "gives way" in its hind-quarters; trembles over the whole body, especially about the flanks and pelvis ; separates itself from the herd ; assumes a staring, expressionless look ; and manifests severe cerebral disturbance. Instead of stupor, attacks of madness and fury may come on ; the animal may bellow, stamp its feet, rear, and dash itself against any object it may encounter, etc. In other cases we may have symptoms of excessive dyspnœa without any apparent changes in the lungs. Gastric attacks may also supervene, as for instance, constipation, slight tympanitis, colic, and bloody diarrhœa, which are very common symptoms of anthrax in cattle, and which sometimes herald in the disease. The urine frequently contains blood (*hæmaturia*). Pregnant cows may abort or be attacked by strong labour-pains. A sanious discharge issues from the natural openings of the body (mouth, nostrils, eyes, anus, and vagina), and death usually ensues in from 12 to 48 hours on account of general weakness and stupor, preceded by convulsions.

Peracute Anthrax (*anthrax acutissimus*) is observed, as a rule, sporadically in very strong individuals, and at the commencement of an outbreak. The patients die either quite suddenly, or in a few hours, with symptoms of cerebral apoplexy or poisoning. Previously healthy animals are sometimes found dead in their stalls in the morning.

Subacute anthrax is comparatively rare, and lasts from 3 to 7 days, or longer. It is distinguished by repeated remissions of fever and considerable emaciation.

The *carbuncles* of anthrax appear in cattle either primarily or during the course of the acute and subacute form, and affect different parts of the skin, such as that of the head, neck, chest, shoulders, abdomen, sheath, udder, flanks, or limbs. They are mostly isolated, circumscribed, or diffuse, and are little, if at all, painful, and then only at the beginning of the attack. They are of a blue-black or dirty dark-red colour, and when cut through are found to be gelatinous or lardaceous, but never suppurating. They become, however, often gangrenous, in which case there is deep gangrene of the skin. Carbuncles in the mucous membrane of the mouth (anthrax of the tongue, anthrax of the palate, and gloss anthrax) show themselves in the form of vesicles and nodes of varying size in the tongue and mucous membrane of the lips, cheeks, and palate with profuse salivation and difficulty

of swallowing. Carbuncles on the mucous membrane of the rectum cause great straining during defecation, with swelling and prolapse of the mucous membrane, accompanied by a bloody discharge. In most cases the cause of this implication of the rectum is injury inflicted during back-raking.

Differential Diagnosis of Bovine Anthrax.—The diseases for which anthrax is most frequently mistaken are: poisoning, inflammation of the brain, cerebral apoplexy, pulmonary apoplexy, heat apoplexy, death by lightning, rabies, gastro-intestinal inflammation, leucæmia, foot-and-mouth disease, and quarter-ill. As the course of the disease is very rapid, it can often be recognised only, *post-mortem*, by finding the bacilli and by employing test inoculations. With reference to the differentiation of quarter-ill, see Vol. II., p. 293.

(2.) *Anthrax in Horses.*

Pathogenesis.—Many of the reports on anthrax in horses are unreliable; because in them petechial fever has been frequently mistaken for anthrax. The disease generally occurs in anthrax districts, where nearly all the domestic animals may become infected. The absorption of anthrax spores or bacilli by means of the fodder is consequently regarded as the usual cause. Anthrax is very common among the horses of Asiatic and European Russia, where it occurs chiefly in swampy districts which are exposed to inundations, such as the low-lying countries of the Volga, where it is known as "Siberian plague" (Haupt and Blumberg). In German South-West African protected territories it goes under the name of "*Bloedziekte*," *i.e.*, blood disease (Sander). The infection may be communicated along with the stings of flies. The disease may be transferred indirectly from cattle or sheep to horses in other ways, as for instance, by putting on harness of untanned infected leather (Bobertag).

The contagium may be introduced into perfectly healthy districts and stables by imported oats or hay, which theory is the only explanation that can account for the rare sporadic cases of anthrax in the army. In the whole Prussian army only 15 horses were affected with, and died from, anthrax in the years 1889 to 1894.

Symptoms.—Anthrax occurs among horses usually in the acute or subacute form. The first symptom is high fever, rang-

ing from 103° to 107° F., or more, with a very small and finally imperceptible pulse of from 80 to 100 per minute. The rise in temperature is often manifested by rigors and muscular spasms. The external temperature of the body is very unequally distributed. The mucous membranes of the head, especially the conjunctiva, are cyanotic, and sometimes slightly yellow. Lachrymation of both eyes is often present, and the mental faculties are exceedingly torpid. The animal has a dull, stupid look, is apathetic, appears stunned, and staggers when walking; or there are symptoms of inflammation of the brain (excitement, restlessness, and convulsions). Colic, which frequently appears at the beginning of the attack, is a regular and very characteristic symptom of anthrax in horses. It is seldom very severe, and is followed by watery dysenteric diarrhœa. As in cattle, the respiration is exceedingly accelerated and difficult. In cases of carbuncular swellings of the mucous membrane of the pharynx and the larynx, we find symptoms of angina such as salivation, difficulty in swallowing, large swellings in the region of the larynx and the pharynx, dyspnœa, and even suffocation. Death takes place with increasing weakness, profuse perspiration, etc., in from 6 to 30 hours, on an average. In a few cases the disease may run a fatal course in from a quarter to half-an-hour. Recoveries are very rare.

Carbuncles on the skin occur chiefly on the hypogastrium, lower part of the breast, inner surface of the fore-quarters and hind-quarters, scrotum, vulva, etc. The swellings of the hind-quarters cause lameness. This form of anthrax runs a course of 2 or 3 days. Gloss anthrax, namely, carbuncles in the mucous membrane of the tongue, have been found on rare occasions in horses (Gresswell).

[For convenience sake, we may divide equine anthrax into anthrax accompanied by swelling of the throat, and anthrax without this symptom. In the former, the disease seems to be located chiefly in the organs of breathing; in the latter, in those of the abdomen. The one might be termed the thoracic or respiratory form; the other, the abdominal. Fred. Smith (*Veterinarian*, February and March, 1897) states that the latter is about 4 times as common as the former in horses. In the cases I have seen, principally in Assam and Cachar (India), the respiratory variety was the more frequent. As addenda to the symptoms described in the text, I may state that the animal suffers from depression of spirits and stupor. The breathing (in the thoracic form), which is almost entirely abdominal, as may be seen by the heaving of the flanks, becomes more and more hurried, until at last the animal falls from exhaustion, becomes convulsed, and dies, apparently from suffocation. There is sometimes a flow of a rusty-coloured fluid from the nostrils, and often,

towards the end, and after death, a discharge from them of foam more or less tinged with blood. I have never seen the disease kill in less than 3 or 4 hours from the first appearance of the symptoms. The course of the malady may run on for 6 or 7 days. The mortality from anthrax in India is probably about 80 per cent. When there is swelling of the neck, the nose is poked out, and the neck (especially about the region of the throat) and intermaxillary space become swollen, often to an enormous extent, so that the head and neck become hard and immovable, as if they were cut out of a single piece of wood. The amount of the swelling, however, greatly varies.

In the abdominal form of anthrax there is sometimes prolapse of the rectum; and, in a few cases, serous tumours form about the flanks, underpart of the belly, and scrotum.

To state the matter generally, the symptoms of anthrax are those of impeded respiration and of infiltration into various tissues. Hence, the painful breathing, the interference with the functions of the brain, the semi-paralysed condition of the patient, and the local swellings.—TR.]

Differential Diagnosis in Horses.—Anthrax is distinguished chiefly by its stormy and feverish course, and by the variety and rapid changes of the different symptoms, which never appear in regular succession. It is confused most frequently with petechial fever, and next to that with colic, cerebral apoplexy, œdema of the lungs and septicæmia. As we have already mentioned when discussing petechial fever, this disease was frequently, even in recent times, regarded as a form of anthrax, which view is altogether wrong; for the bacilli of anthrax have not been found in the blood of the animals affected with petechial fever; and all attempts to transmit petechial fever, either by inoculation or by infection, have failed. A correct diagnosis of anthrax can be obtained only by the demonstration of the bacilli, and by transmitting the disease to a rabbit or a sheep by inoculation. The adoption of inoculation is also indicated by the fact that, according to recent observations, the gelatinous capsule of the anthrax bacilli of the horse appears to be more difficult of demonstration than in case of cattle (Schmidt).

Remittent Anthrax.—A peculiar remittent course of anthrax in the horse has been observed by Burke in a series of horses in India. The temperature in those cases fell to normal, or even below, with complete disappearance of all symptoms of sickness, so that the animals in such cases were quite convalescent; whereon, nevertheless, a new attack followed in a few days, and the animals at last died of exhaustion. The whole course of the disease forcibly reminded one of the relapsing fever of human beings. The often very rapid emaciation which appeared on the first attack, even within a few hours, was quite characteristic; so also was the

consecutive development of a paralytic condition of various groups of muscles.

[On account of the hurried breathing and injected condition of the mucous membranes of the nostrils and eyes, anthrax is sometimes mistaken for congestion of the lungs, which can be distinguished by the fact that the patient fights for breath, and does not exhibit the depression and semi-unconsciousness of a horse suffering from anthrax. Besides, the characteristic swelling of the neck is absent.

The disease which, above all others, clinically resembles equine anthrax is South African Horse Distemper (Vol. II., p. 652), especially when the attack of anthrax takes the respiratory form. The clinical resemblance between the two is greatly heightened by the fact that the symptom of swelling of the head, intermaxillary space and neck is common to both. Sander (Vol. II., p. 652) is by no means alone in the mistake he made; for many English veterinary surgeons who have sojourned in South Africa have fallen into the same error. In fact the question was not finally settled until Edington had published the results of his bacteriological researches on the South African malady. Here, the most salient points in *differential diagnosis* are: Influence of time of the year on Horse Distemper. (2) Influence of dew on the development of the South African micro-organism. (3) Protection against Horse Distemper by restriction in diet to dry fodder. (4) Difference in the character of the nasal discharge; that of Horse Distemper being generally white and very frothy, with an extremely copious flow of liquor sanguinis. (5) Charged condition, in Horse Distemper, of the bronchial tubes with fluid. (6) Difference in the *post-mortem* appearance of the blood. (7) Unchanged condition of the spleen in Horse Distemper. (8) The fact that Horse Distemper is peculiar to the equidæ. (9) Absence of the bacillus anthracis in Horse Distemper.—TR.]

(3.) *Anthrax in Sheep and Goats.*

Pathogenesis.—Gerlach was the first to recognise as anthrax the disease of sheep which was formerly known as "apoplexy." It appears to be almost always due to fodder. Stings of flies, injuries of the skin during shearing (Nocard), utilisation of gravel-pits, in which the dead bodies of victims of anthrax had for 10 or 12 years previously been buried, etc., may in some cases be the direct or indirect cause of the disease.

Symptoms.—Anthrax attacks *sheep* generally in the apoplectic form (*anthrax acutissimus*). The animal appears as if suddenly stricken with apoplexy, staggers, falls down, is seized with spasms and convulsions, and dies in a few minutes with a discharge of black blood from the natural openings of the body, and is often found dead in the stall in the morning. The course of acute anthrax lasts somewhat longer, although generally

only from 30 minutes to 2 hours. We sometimes find symptoms of congestion of the brain, such as excitement, restlessness, running to and fro, staggering, etc., and at other times, symptoms of congestion of the lungs, namely, very accelerated breathing, frequent pulse, palpitation of the heart, cyanosis of the mucous membranes, bloody discharge from the natural openings of the body, etc.

Subacute anthrax is very rare in sheep. It is sometimes ushered in by premonitory symptoms, especially by digestive and intestinal troubles of an inflammatory nature. Frequent and strenuous attempts to dung, with constant whisking of the tail, is a characteristic symptom. Carbuncles are seen very rarely on the head, throat, or udder.

The course of anthrax in *goats* is similar to that in sheep; but is not so severe as a rule.

Differential Diagnosis.—Anthrax* in sheep has been confused most frequently with malignant œdema. The affections which Haubner and others described as anthrax, and which were characterised by crackling swellings of the hind limbs, were certainly malignant œdema.† In cases of doubt, Cadiot and Ries recommend that pressure should be made on the nose of the patient with the fingers for a few seconds, to excite urination. It is stated that hæmaturia is always present in anthrax.

(4.) *Anthrax in Pigs.*

Pathogenesis.—Cases of spontaneous anthrax in pigs are rare. Swine erysipelas (Vol. II., p. 225), which was formerly almost universally regarded as a variety of anthrax, has nothing to do with it. Consequently, the literary reports of former times on anthrax in pigs are of little value. Pigs are more or less immune to anthrax, which can be transmitted to them only with great difficulty. In fact, the inoculation experiments of Brauell, Renault, Toussaint, Arloing, Cornevin, Thomas, Schindelka, von Ratz, and others, have been attended with negative results. True cases of anthrax seem to occur among pigs only during widely-spread anthrax epizoötics and in anthrax districts, or

* Anthrax in sheep is often confused with braxy and louping-ill.—Tr.

† With respect to this statement, *The Journal of Comp. Path. and Therap.*, Sept., 1898, remarks that "it is far more probable that the cases referred to were quarter-evil."—Tr.

after eating the flesh of anthrax-infected animals,* in which case local affections in the pharynx and mouth usually appear. These remarks are confirmed by the recent experiments of Crookshank, who has proved that anthrax can be produced in both old and young pigs by feeding with portions of the dead bodies of anthrax-infected animals. Trombitas saw 14 pigs die of anthrax within 6 days, after having been fed on the flesh of an anthrax-infected cow. Von Ratz observed in pigs of a Polish and Chinese cross that the anthrax infection took place at the beginning of the digestive canal, most probably from the follicles of the tonsils. According to his experience, the power of resistance to anthrax varies in different breeds of pigs. Crookshank and Perroncito succeeded in experimentally producing the disease by the injection of virulent blood and of bacilli cultures. Tschernogoroff failed to produce inoculation anthrax among 16 pigs by feeding them with portions of organs affected with the disease, and with other materials containing the spores. On the other hand, 6 out of 33 pigs sickened of the disease after subcutaneous inoculation of the same virus.

Symptoms.—Anthrax angina is characterised by carbuncles on the mucous membrane of the pharynx and larynx. The animals are very feverish, and the intermaxillary space is greatly swollen. This swelling may spread along the course of the trachea to the lower part of the chest and the inner surface of the fore-quarters, and thus give rise to symptoms of angina of the pharynx and larynx, such as salivation, retching, stiff attitude of the head and neck, hoarseness, difficulty in swallowing, regurgitation, vomiting, cyanosis of the mucous membrane of the mouth, dyspnœa, panting, harsh breathing, etc. The skin is stained with blood, and the animal shows symptoms of paralysis. Death takes place usually from suffocation. In anthrax of the tongue and palate, clear vesicles, which subsequently become purple and even black, are seen on the mucous membrane of the tongue, palate, lips, etc. Zschokke has described in pigs the occurrence of carbuncles on the skin of the back.

* McFadyean reports an outbreak in which about 14 pigs were attacked with anthrax, probably from eating the flesh of an anthrax-stricken heifer. In all of these pigs, the most prominent symptom was swelling in the region of the throat. McFadyean states that "swelling of the throat in the course of an attack of acute illness is in the pig almost pathognomonic of anthrax."—Tr.

The anatomical changes in anthrax of pigs consist in a gelatinous, hæmorrhagic infiltration of the parts surrounding the pharynx; hæmorrhagic swelling of the lymph glands, spleen and liver (according to Crookshank, the spleen may be normal); and hyperæmia of the other organs. In some cases, enteritis with submucous and subserous hæmorrhages is present. Although the spleen and blood contain only a few anthrax bacilli, they are rich in putrefactive bacteria, the presence of which greatly increases the difficulty of the microscopical demonstration of the anthrax bacilli, and transmission by inoculation (Crookshank).

(5.) *Anthrax in Dogs and Cats.*

Our knowledge of anthrax in dogs is very defective. According to Straus, age confers on them comparative immunity. This observer states that newly-born dogs are extremely susceptible to anthrax when inoculated with moderately virulent material. Bardach inoculated anthrax intravenously in 25 dogs from which the spleen had been removed, and in 25 normal dogs. Of the former 19 died of anthrax; of the latter only 5. The eating of meat infected by anthrax seems to be the most frequent cause of the disease in these animals. On this account, anthrax occurs in dogs usually in the intestinal form and in that of local anthrax of the mouth and pharynx. Cornevin saw 5 dogs out of 7, which had been eating parts of the dead body of an anthrax-infected cow, die on the evening of the same day. Peuch saw a case of anthrax from feeding on infected flesh in a dog which had a wound on the tip of its tongue. Some supposed instances of anthrax in dogs are probably nothing else than cases of ptomaine poisoning, which is a disease that was formerly frequently confused with anthrax in man.

Cats, lions, and other carnivora may become infected with anthrax in a similar way to dogs.

(6.) *Anthrax in Birds.*

Pathogenesis.—Pasteur states that birds are immune to anthrax on account of the high temperature of their blood; but that they may be made susceptible by reducing their bodily heat. Pasteur's statements have lately been confirmed by Wagner, who found that this immunity is lost by the introduction of certain chemical agents (chloral hydrate and antipyrin)

which have a weakening effect on the birds. Czaplewski also found in his experiments that only very young pigeons of certain breeds are susceptible to anthrax. Koch, Gaffky, Löffler, Perroncito, Hess, Kitt, and others, obtained negative results with their inoculation experiments in birds. On the other hand, the inoculation experiments of Feser and Œmler have been successful in several cases. Œmler observed in his experiments that small birds (robins, finches, goldfinches, canaries, sparrows, yellow-hammers, etc.), and especially young birds, can be very easily infected; that the larger birds are less susceptible; and that only birds of prey are quite immune. Usually birds do not become infected, except during a general anthrax epizoötic, and after feeding on flesh and blood of anthrax cadavers. Farmyard poultry may also become infected.

Symptoms.—The course of anthrax in birds is exceedingly rapid and severe, and may terminate suddenly or after a few hours' suffering. Towards the end, the bird staggers and trembles, or becomes violently convulsed, and dies with a bloody discharge from the mouth, nostrils, and anus. In other cases the disease lasts for about a day. The bird is depressed and weak, its feathers are ruffled, it droops its wings, and suffers from dyspnœa and bloody diarrhœa, and the mucous membranes and corpora cavernosa (comb and wattles) are livid. Carbuncles are comparatively frequent, and appear chiefly on the comb, wattles, conjunctiva, tongue, palate, extremities, webbed portion of the feet, etc. Mistakes are often made between anthrax and chicken cholera, cerebral apoplexy, and malignant œdema.

Anthrax in Man is chiefly found in knackers, butchers, tanners, herdsmen and veterinary surgeons, in consequence of an infection acquired whilst cutting up the cadavers, and it generally assumes the form of carbuncles (malignant pustule) on the arms, face, neck, etc. The appearance of the pustule is followed by fever, and usually by death. Intestinal anthrax mostly occurs from eating infected flesh. The treatment of the malignant pustule consists in its destruction by the hot iron, excision or by the use of strong disinfectants, such as corrosive sublimate, creolin and carbolic acid.

RABIES (*Hydrophobia*).

Rabies in general—Canine rabies—Symptoms of rabies in man—Bovine rabies—Equine rabies—Rabies in cats—Rabies in pigs—Rabies of sheep and goats—Rabies of birds.

A.—RABIES IN GENERAL.

History.—Rabies is one of the oldest known diseases of animals. In the fourth century, B.C., it was described by Aristotle, who writes: "Dogs suffer from madness which puts them in a state of fury, and all the animals that they bite, when in this condition, become also attacked by rabies." We find allusions to it in the works of Virgil, Horace, Ovid, and Plutarch. Cornelius Celsus, who lived in the first century of the Christian era, was the first to allude to human rabies, and to employ the word "hydrophobia." Dioscorides recommends excision of the wound as a protective remedy; and Galen in the second century gives special remedies for rabies. Among the older authors we may cite Plinius the younger, Columella, and C. Aurelianus. No allusion to rabies is to be found in the literature of the Middle Ages. Bauhin, in 1591, mentions in his "*Memorabilis historia luporum aliquot rabidorum*" (the transmission of rabies by wolves to man). In 1604 an epizoötic of rabies broke out in Paris (Andry); towards the end of the seventeenth century, in Italy (Baglio, Rammazzini); in 1708, through Swabia (Camerarius and Scharff); from 1719 to 1723, in France and Germany; from 1754 to 1760, in England; and from 1779 to 1807, in America, especially through the West Indies and Peru. Towards the end of the eighteenth, and at the beginning of the nineteenth century, rabies spread over the whole of Europe, and many rewards were offered during that time for the discovery of a remedy for it. The names of Chabert and Hunter should be kept in remembrance for the good work they did in the investigation of this disease. An epizoötic of rabies raged amongst foxes from 1803 to 1830 in Southern Germany and Switzerland (Köchlin and Franque). During 1814 and 1815, Viborg in Copenhagen and Waldinger in Vienna improved the methods of the experimental investigation of rabies. In 1817 and 1818, Delabère-Blaine and Greve in England greatly enriched the clinical knowledge of this malady. Rabies was prevalent during 1822 in Holland; during 1823 and 1824 in Berlin; and during 1824 in Sweden

and Russia. In 1828, during the epizoötic which raged from 1823 to 1830, Hertwig published his "Contributions to the better understanding of Rabies," which is a report of a great number of carefully-executed experiments on the transmission of rabies, and which greatly advanced the knowledge of this disease. In 1832 Youatt in England and Prinz published their works on rabies. In the thirties the disease was very common in Prussia; and from 1838 to 1843 in Austria and Würtemberg. During 1852 and 1853 Prussia was again visited by this scourge. In 1853, 150 dogs suffering from rabies were taken to the canine hospital of the veterinary college in Berlin, and 267 cases occurred in Hamburg. During 1861 rabies broke out as an epizoötic in the Rhenish countries and in France; and from 1863 to 1871 in Würtemberg, where 597 animals became infected, and 449 people were bitten, of whom 23 died. From 1862 to 1867, and again from 1873 to 1876, it raged in Vienna; from 1865 to 1866 in Saxony; and from 1871 to 1876 in Saxony, Bavaria, and Prussia. Since the introduction of the Imperial laws respecting epizoötics, the number of cases of rabies has very considerably decreased in Germany.

Erroneous opinions about the nature of rabies have been held for a long time. In spite of the proved infectious character of the disease, it was formerly believed by the majority of people that rabies could originate from the respective influences of great heat, unsatisfied sexual desire, thirst, nervous excitement, anger, jealousy, too high feeding, etc. Virchow and others called attention as early as 1854 to the error of this supposition. It is only during the last few decades that the exclusively infective nature of the disease has been generally accepted. In the year 1881 Pasteur greatly increased our knowledge of the nature of this disease by his work in the field of protective inoculation, and gave a permanent impulse to further experimental researches.

Etiology.—The infective matter of rabies has not yet been produced in a pure condition in spite of the labours of numerous investigators, such as Raynaud, Lannelongue, Gibier, Fol, Babes, Pasteur, Koch, and others. Pasteur, however, has shown that the virus is purest in the central nervous system (brain and spinal cord) of infected animals, and less so in the peripheral nerves, salivary glands, lachrymal glands, aqueous humour of the eye, pancreas, mammæ, testicles, kidneys, and in their secretions. The nervous system appears to offer the

most favourable condition for the development of the virus. The blood does not contain the infective material. At least all recent experiments made on the transmission of rabies to healthy animals by means of the inoculation or transfusion of the blood of rabid ones have proved negative (Renault, Galtier, Paul Bert and others). The lymph, lymphatic glands, and flesh are also free from the virus. The contagium is at all events fixed, not volatile; and is purely endogenous, not ectogenous; that is to say, the animal body is absolutely necessary for its development. Paul Bert proved that it is a solid body, by filtering the saliva of rabid dogs through plaster of Paris plates and by then showing that the filtrate was innocuous. Pasteur found microscopically in the brains of rabid animals fine granules in the form of extremely minute points which could be stained with aniline solutions, and which he regarded as very minute micro-organisms. He did not succeed in cultivating these bodies, which he considered to be neither micrococci nor bacilli. Negri has recently discovered the causative agent in rabies, in the shape of protozoa of various forms 1 to 27 μ in diameter, which are situated in various parts of the brain, especially in the cornu Ammonis. This discovery has been confirmed in various directions, and will possibly attain to a high degree of diagnostic value (Beck). The 1 to 27 μ Negri bodies cannot, however, be the cause of the infection; because the virus of rabies may be filtered, even through filters of specially fine construction (Schüder).

The recent investigations of Babes show that the existence of a characteristic micro-organism of rabies cannot be proved even by our most modern bacteriological methods. These investigations, however, point to the probability of our being able to cultivate through several generations, from the central nervous system of rabid animals, an unknown substance which possesses the capability of producing rabies, and behaves to external agents, as a rule, like a bacterium; although it is much more resistant to carbolic acid than any known bacterium. Babes cultivated this substance and produced rabies in dogs, rabbits, guinea-pigs, cats, rats, and mice by inoculating it into the vitreous humour of the eye, or between the cerebral membranes by trephining. Bruschettini professes to have found the cause of rabies in a bacillus; Memmo, Grigorjew, and Levy consider it to be a blastomycete. Anrep obtained from the brain of rabid rabbits an exceedingly virulent material which appeared to be of the nature of an alkaloid.

The vitality of the contagium of rabies seems to be greater than was formerly supposed. The inoculation experiments carried out by Hertwig by means of the saliva and blood of

rabid dogs gave negative results 24 hours after their death Gibier, on the contrary, found that the saliva remained virulent for 24 hours, and Mergel observed that the brain continued virulent for 15 days. Similar results were obtained in Dorpat. Pasteur preserved the brain of rabid dogs for 3 weeks under a very low temperature (10·4° F.) without the infective matter losing its virulence. This organ, if kept in moist carbolic gauze, retains the contagium in an active condition for months. The infective matter remained virulent for 3 or 4 weeks during summer, when kept in tubes which had their ends closed by fusion. Pasteur proved that the virus can retain its power of infection for 4 or 5 days when exposed to far advanced putrefaction. It was formerly supposed that it lost its infective power directly after putrefaction set in, and consequently soon after the dead body had cooled down, that is to say, within 24 hours after death. Galtier found that, in spite of putrefaction, buried cadavers remained virulent for from 15 to 44 days. According to Viala, the contagium of rabies continues active for over 5 months when kept cool and in a vacuum. Bruschettini states that it becomes inert after having been kept in carbonic acid for 13 days.

In all cases, the transmission of the infective matter to other animals appears to be directly effected by the bite of a rabid animal without any intermediate bearer. Rabies is therefore to be looked upon as an inoculation disease, the saliva serving as the vehicle for the contagium. Roux and Nocard have pointed out the very important fact that 2 or even 3 days before the appearance of rabies, the saliva contains the contagium, and is consequently virulent towards the end of the period of incubation. It is very doubtful if the virus can be absorbed in the digestive canal by the consumption of the flesh, milk, or saliva of rabid animals, as Galtier states was the case with rabid saliva in his experiments with rabbits. We may be quite certain that the ingestion of the flesh or milk of rabid animals has never produced any injurious consequences. As a proof of this fact we may mention that Nocard fed a young fox for 2 months on the brain and spinal cord of 12 rabid dogs, without any ill result to the fox. Wirchikowski states that gastric juice destroys the contagium of rabies. On the other hand, we have to point out that the intra-cranial inoculation of the milk of rabid animals has produced rabies (Nocard, Roux, and Bardach). Perroncito and Carita produced rabies in guinea-pigs by in-

oculation with the medulla of a young rabbit, the mother of which had become rabid by inoculation. Callignac and Gibier state that they have observed hereditary transmission of rabies. The experiments of Renault, Roux, Galtier, Zagari, Bombicci, and others on this point have, on the contrary, given negative results. In any case, the hereditary transmission of rabies is certainly very rare, and cannot take place through the placental circulation, as the blood does not contain the agent of infection in rabies.

The Behaviour of the Virus of Rabies with Various Agents.—According to the investigations of De Blasi and Russo-Travoli, the rabic virus is destroyed by a 1 per cent. solution of creolin and by lemon-juice in 3 minutes; by Ceylon cinnamon, a 2 per cent. solution of *liquor ferri*, a 5 per cent. solution of hydrochloric acid and salicylic acid, and a 50 per cent. solution of nitrate of silver, in 5 minutes; by sulphuric acid and liquor ammoniæ in 10 minutes; by a 4 per cent. solution of boric acid in 15 minutes; by a 1 per cent. solution of permanganate of potash in 20 minutes; by a 5 per cent. solution of carbolic acid in 50 minutes; by a 3 per cent. solution of carbolic acid in 60 minutes; and by a 2 per cent. solution of carbolic acid in 120 minutes. According to this, creolin is the strongest and carbolic acid the weakest disinfectant for the virus of rabies. We learn from the experiments of Celli that the virus of rabies is rendered inert by hot steam in half-an-hour; by a temperature of from 122° to 140° F. in 1 hour; by one of 113° F. in 24 hours; by corrosive sublimate (1 to 100,000), permanganate of potash (from 2 to 5 per cent.) and alcohol (50 to 90 per cent.) in 24 hours; by a 25 per cent. solution of alcohol in 5 days; and by a 15 per cent. solution of alcohol in 7 days. The emulsion of the brain substance instantly loses its virulence when it is slightly acidulated with one or two drops of acetic acid, or when it is made slightly alkaline with soda. According to Frantzius, the Röntgen rays weaken the poison of rabies; Von Ratz found that decomposition also effected this result. On the other hand, the mitigation of the contagium by repeated transmission through the bodies of goats, which was asserted by Pourtalé, does not occur according to the researches of Nocard. Finally, the bile of rabid animals possesses an antitoxic influence on the contagium of rabies (Frantzius, Kraus, Salomon).

Pathogenesis.—If the virus contained in the saliva of a rabid animal penetrates the skin through a bite, it may remain for a long time at the site of the bite, or it may enter sooner or later into the body by means of the blood or along the nerve-tracts. According to the most recent investigations, it appears that the virus of rabies moves from the bitten part chiefly within the nerve-tracts in a centripetal direction to the central nervous system, possibly enclosed in migratory cells (Duboué, Di Vestea,

and Zagari). It is evident that the virus becomes best developed in the brain and spinal cord; the former being the principal seat for the development of furious madness; the latter for that of dumb madness. Inoculation experiments have shown that the disease breaks out most rapidly from direct inoculation into the brain, under the dura mater, and that the period of incubation is proportionate to the distance of the site of the inoculation from the brain.

The *period of incubation* is longer than in other infective diseases, and in dogs amounts on an average from 3 to 6 weeks, with a maximum of several months, and a minimum of only a few days. [Hunting puts the usual period at about 25 days.—TR.] The virus may remain for a long time (as we have already said) at the site of the bite and may become absorbed only in small quantities at a time; or it may rapidly, and in large quantities, penetrate into the body. According to Protopopoff, the younger the animal, the shorter is the period of incubation.

Occurrence.—The following species of domestic animals are liable to be affected with rabies: dogs, cats, cattle, horses, asses, mules, jennets, sheep, goats, poultry, and pigeons; so are wolves, foxes, jackals, hyænas, badgers, martens, monkeys, stags, roes, antelopes, skunks, rabbits, guinea-pigs, rats and mice. Human beings, also, are often affected. It, however, occurs most frequently among dogs. Out of all the supposed predisposing causes—such as breed, age, sex, climate, country, etc.—the season of the year is the only one which has any influence. As in other infective diseases, the number of cases of rabies is greater in summer than at other times. Some animals appear to possess a certain individual immunity to it. The disease is naturally most frequent in places where there is a large number of dogs, and where there is much traffic, as in large towns, especially when the protective police measures are defective. On account of the disease being transmitted from dogs to wolves, and *vice versâ*, it is frequently enzoötic in the neighbourhood of mountains and plains in which wolves are found, as in the Vosges, Carpathian Mountains, Russia, etc.

Statistics.—In the German empire a total of 12,000 animals (dogs, cats, horses, cattle, sheep, pigs and goats) became rabid from 1886 to 1902. The districts of the empire which are most affected are: Gumbinnen, Bromberg, Königsberg, Posen, Marienwerder, Oppeln, Liegnitz, Breslau; also the Russian-Polish and the Austrian borders. The king-

dom of Saxony, too, is sometimes greatly affected. In Germany, on an average, about 700 animals annually become rabid; of which from 500 to 600 are dogs. The figures for the individual years are as follows :—

Year.	Total number.	Dogs.	Cattle.	Horses.	Sheep.	Pigs.	Cats.	Goats.
1886 . . .	577	438	92	5	32	7	3	1
1887 . .	556	423	99	6	6	17	4	1
1888 . . .	548	397	101	7	17	18	5	2
1889 . . .	493	410	65	5	3	6	4	—
1890 . . .	714	590	98	4	2	9	11	—
1891 . . .	543	445	70	11	8	4	3	1
1892 . . .	500	387	69	8	7	27	2	—
1893 . . .	466	410	39	3	7	4	3	—
1894 . . .	557	471	73	4	—	6	3	—
1895 . . .	489	431	35	4	8	8	1	2
1896 . . .	939	724	190	8	6	8	2	1
1897 . . .	905	770	106	11	3	4	8	3
1898 . . .	1,202	904	223	14	44	5	9	3
1899 . . .	1,154	911	171	9	38	17	7	—
1900 . . .	987	798	153	6	—	13	10	5
1901 . . .	676	500	78	6	5	22	4	1
1902 . . .	612	516	77	3	2	7	6	1

[South Africa is practically free from rabies. The only case reported for many years was one of a dog which was found to be rabid in Port Elizabeth, by Britton, in 1894.]—Tr.

Anatomical Changes in Rabies.—In contrast to other infective diseases, the changes which are found on *post-mortem* in rabid animals are neither constant nor specific; in fact they are characterised more or less by negative appearances. The general changes, which are in no way typical, are as follows : The very emaciated cadavers become rapidly putrid, and, in the case of the larger domestic animals, greatly distended by gas in the hind parts of the body. The blood is thick and of a dark-red colour; the muscles appear granular and affected with fatty degeneration, and the heart, liver, and kidneys show parenchymatous degeneration. The mucous membrane of the mouth is congested and swollen, chiefly at the base of the tongue; the tonsils are enlarged and inflammatorily infiltrated; and the salivary glands are hyperæmic. The mucous membrane of the pharynx and larynx is reddened, swollen, and even studded with small hæmorrhages. Various kinds of foreign bodies are sometimes found in the pharynx and œsophagus. The stomach frequently contains straw, hair, feathers, pebbles, pieces of brick, particles of wood, bits of leather, portions of whipcord, and other foreign and indigestible

substances ; but little or no food. Wortley Axe found foreign substances in the stomach of 90 per cent. of 200 rabid dogs which he examined on *post-mortem* ; but no trace of food. He considers that this condition of the stomach is the most important feature of the whole autopsy. Galtier found foreign bodies in the stomach in from 50 to 70 per cent. of 300 rabid dogs of which he made autopsies. The mucous membrane of the stomach is congested and swollen ; and small hæmorrhages and hæmorrhagic erosions are frequently seen on the surface of its folds. The intestine is generally empty, and its mucous membrane and the mesenteric glands are congested and swollen. The spleen is hyperæmic, swollen, and sometimes infiltrated with hæmorrhagic tumours. We find in the urine, albumen and sugar ; and in the case of dogs, biliary pigments. The respiratory mucous membrane is purple and the lungs are full of blood. The changes which are found in the brain and the spinal medulla are very inconstant. Sometimes we find hyperæmia, œdema and extremely minute hæmorrhages ; but at other times no changes are apparent.

Microscopic Conditions.—Csokor and others state that a considerable accumulation of lymph corpuscles may often be seen with the microscope along the capillaries and within the walls of the vessels and in the grey matter. The vessels of the grey matter are dilated and show microscopically small hæmorrhages and hyaline thrombi. Babes advises the location, in the medulla oblongata of rabid dogs, of a constant chromatolysis of nerve-cells, emigration of embryonic cells, brown nuclei and hyaline corpuscles. Thrombosis of vessels and minute hæmorrhages ; the "madness-nodules" or "rabies tubercles" (nodules rabiques), that is to say, cellular infiltration and proliferation of cells of a fibroblastic type in the vicinity of the nerve cells, are also of appreciable value in the diagnosis of rabies. Van Gehuchten and Nélis have found that in dogs which had died of madness in the streets, the cerebro-spinal and sympathetic ganglia, especially the plexus nodosus of the vagus, display characteristic changes (enlargement, injection, ecchymosis, much endothelial cell-proliferation of the capsule). The subsequent examinations of Hébrant, Vallée, and others have established the fact that these changes are present in dogs which have died of rabies, but, on the other hand, show that they may be absent in rabid dogs which have been killed at an earlier stage. Bohl found them also in other diseases, *e.g.*, distemper. The histological diagnosis is, accordingly, not more reliable than the dissection.

Prognosis.—Pasteur states that he has observed in his inoculation experiments with dogs, a few rare cases of recovery after the commencement of the first symptoms of the disease ; but only when the attack was slight. Trasbot, Janson, Szpil-

mann, Silbermann, Bouley, Decroix and Menecier have seen a few recoveries in rabid dogs. Rabies must, however, be regarded as an almost invariably fatal disease.

The inoculation mortality is not so great. Hertwig found that only 37 per cent. of the animals inoculated by him became infected; and Renault 67 per cent. The comparative smallness of these figures must be due to the fact that in inoculations, the infective matter frequently does not adhere to the inoculation wound; that it may remain ineffective on the hair or on the surface of the skin; or it may be washed away by the hæmorrhage caused by the inoculation. In the same way we may account for the fact that the bite of a rabid animal does not invariably produce rabies. It seems that on an average only from 20 to 30 per cent. of those bitten by rabid animals become infected. Hertwig puts the percentage at 5; Haubner, at 40; the Veterinary College of Lyons, at 20; and that of Alfort, at 33. Röll calculates that in Austria during the years 1877–1887, the percentage among horses was about 40; among cattle and sheep, 50; among pigs, 36; and among goats, 20.

The percentage of deaths in mankind is considerably influenced by treatment. Bollinger states that of 100 bitten men only from 8 to 47 per cent. become infected. Pasteur puts the percentage at from 16 to 80.

Therapeutics of Rabies.—Treatment is purely prophylactic; for it is of no avail after the appearance of the symptoms of the disease. Although in men, we should always cauterise the wound with the hot iron, caustic potash, sulphuric acid, corrosive sublimate, etc., or excise the bitten part or the cicatrix; we should, for several reasons, apply such means to our domestic animals only in exceptional cases. Prophylaxis consists chiefly in the application of indirect protective measures, of which the best are those of the German police regulations of 23rd June, 1880, with regard to epizoötics. The utility of this law was greatly enhanced by the introduction of a tax on dogs, by which the number of dogs and consequently the number of cases of rabies were decreased, as was statistically proved in Bavaria. In large towns the compulsory wearing of muzzles, combined with preventive treatment of the wounds, and the establishment of a tax on dogs, have proved a most excellent means for checking the spread of rabies, provided of course that the muzzles effectively answer their purpose. In Berlin, for instance, the compulsory wearing of muzzles

has greatly diminished the prevalence of rabies. The following statistics show the benefits to be obtained from these prophylactic measures. In Bavaria (tax on dogs, 1876; law in regard to epizoötics, 1880) the number of rabid dogs decreased from 821 in 1873, to 11 in 1885; in Baden, from 53 in 1874 to *nil* in 1885; in Saxony, from 287 in 1866 to 16 in 1885; and in Prussia, where only the law for regulation of the epizoötic was in force, from 672 in 1878 to 352 in 1885.

[In Great Britain, the regulations against rabies, which were first put in force in 1886, have proved singularly ineffective, considering that this disease is communicated only by the bite of rabid animals. The statistics of canine cases are as follows: In the year 1887 (217 cases); 1888 (160); 1889 (312); 1890 (129); 1891 (79); 1892 (38); 1893 (93); 1894 (248); 1895 (672); 1896 (438); and in 1897 (248). The faulty regulation of rabies in Ireland has long been a serious danger to life and property in Great Britain as well as in Ireland. The good effect of the muzzling order of 1896, despite its partial application, is shown by the following table of canine rabid cases:—

Month.	London. (Muzzling order put into force 7th Feb.)	Surrey. (Muzzling order put into force the whole year.)	Middlesex. (Muzzling order put into force 17th Feb.)
January	20	15	11
February	25	12	9
March	22	11	4
April	11	7	5
May	11	3	5
June	12	3	3
July	14	4	3
August	2	2	1
September	1	2	1
October	4	2	2
November	2	1	2
December	4	2	2

As suggested by Hunting, the muzzling act, to be reasonably effective should be put in force—immediately a case of canine rabies is reported—throughout the surrounding country, say, for a radius of 10 to 20 miles; and should be continued in that area for a term sufficient to cover a fairly long period of incubation, say, for not less than 3 months. During an outbreak, every wandering dog which has been taken up by the police, if not destroyed in the meantime, should be segregated in a separate cage for at least 8 days (see Vol. II., p. 580), so as to make certain that the wandering was not due to the effects of rabies. Until Ireland gets a clean bill of health as regards this disease, the importation of dogs from it into Great Britain should be allowed only under quarantine rules similar to those at present in operation with respect to foreign countries. Such importation, except in the case of practically rabies-free countries such as South Africa and Australia,

remains a constant danger to the United Kingdom, and should consequently be permitted only under a quarantine of at least 3 months' duration. It should be remembered that rabies is particularly frequent in Russia. The registration of all dogs in the United Kingdom would be a very desirable enactment, especially, if at the same time a rigid scrutiny into claims for exemption from the dog-tax on the plea that the animals are required for herding purposes, was instituted. At present a large number of dogs escape this tax, solely because they belong to sheep-dog breeds (collie or bob-tailed) and are owned by country people; although they are never employed with farm stock. For valuable suggestions by Hunting and others, regarding the prophylaxis of rabies, see *Veterinarian* for July, 1897.—TR.]

Pasteur's Protective Inoculation against Rabies.—Pasteur stated in 1884 that the virus of rabies could be gradually weakened, by carrying it from dogs to monkeys and by repeated transmissions from monkeys to monkeys. He also pointed out that the virus thus weakened, when injected under the skin or under the dura mater, after trephining the skull, did not produce rabies in dogs, but rendered the inoculated animals immune both to natural and artificial infection. In more recent times, Pasteur published another process of immunising by the use of the dried spinal cord of a rabid animal as the inoculation material. The spinal cord and its membranes are removed from a rabid rabbit under aseptic precautions as far as the cerebellum, and cut into pieces of 2½ inches in length. These pieces are tied to threads, which are suspended in bottles; and the bottles are filled up to a height of ½ inch with caustic potash, and closed with cotton-wool plugs. The bottles are then kept at a constant temperature of 68° F. After 3 or 4 days the portions of spinal cord dry up to the form of friable bands, the virulence of which gradually decreases according to the duration of the desiccation. For instance, rabbits become affected in 7 days, after inoculation with material which has been dried for from 24 to 48 hours; in 8 days, when the desiccation has lasted from 3 to 5 days; and in 15 days when it has been continued for about a week. Animals may be made immune to rabies by at first inoculating them with very attenuated material, and then gradually increasing the strength, so that they can finally bear the strongest virus with impunity. These attenuated inoculations sometimes cause the inoculated animals to become affected with a temporary and partial rabies.

Pasteur states that the effect of the virus increases and the period of incubation diminishes, in proportion to the number

of rabbits and guinea-pigs through the bodies of which the disease is transmitted. The strongest virus is prepared as follows: the experimental rabbits having been trephined with a trephine of ¼ inch diameter, a small quantity of the central nervous system of a rabid dog that has just died is injected aseptically under the membranes of the brain with a Pravaz syringe which has its needle bent at a right angle; the result being that dumb rabies breaks out in 15 days. If the injection be made subcutaneously, the development will be slower. When, after this, the virus is transmitted through a series of rabbits, the period of incubation, as we have already said, becomes gradually shorter, namely, 8 days in the twenty-fifth rabbit, and 7 days in the fiftieth rabbit, with which the maximum of virulence is reached. With an inoculation of ordinary fresh virus, the period of incubation is 15 or 16 days.

These statements of Pasteur were tested by a French commission, among whom were those eminent savants—Paul Bert, Bouley, Villemin, Vulpian, and Tisserand. This commission fully confirmed Pasteur's statements, and consequently there is no doubt that the virus of rabies can be either weakened or strengthened.

Pasteur attempted to immunise human beings after they had been bitten, by inoculating them with attenuated virus. He applied this process first to dogs with a positive result. Altogether there have been inoculated from 1886 to 1900 in the Pasteur Institute in Paris more than 23,000 persons (about 1,500 per annum); and many thousands more in the Inoculation Institutions founded by Pasteur in Russia, Hungary, Austria, Italy, etc., in the following manner. At first a piece, ¾ to 1¼ inch long, of the dried spinal cord of a rabbit is rubbed down in sterile broth, and this fluid (emulsion) is injected directly under the skin of the abdomen in a quantity of from 8½ to 12¾ minims. To begin with, the weakest inoculation material is used and then the strength is daily increased until finally a material of the highest virulence is employed. In Berlin, an inoculation institute was opened in 1898; in that year 137 bitten people were treated; in 1899, 384; and in 1900, 332. It is stated that by this process the mortality in rabies has been reduced from 16 to 0.5-2 per cent. (Pasteur, Cantini, Metschnikoff, Ullmann, Bujwid, Bardach, and others). Von Frisch, Bordoni-Uffreduzzi, de Rentzi, Högyes and other authorities declare that their researches prove that no benefit

can be derived from Pasteur's supposed curative method. Consequently it is impossible at present to form a definite conclusion on this subject.* Even admitting the efficacy of Pasteur's method of curative inoculation, we must not overlook the following points: First of all, we do not know how many of the bitten people were really infected; the percentage being, according to Bollinger, only from 8 to 47. Pasteur puts the minimum at 16 per cent. Also, we cannot tell, in many cases, whether the bite has been inflicted by a rabid animal or by one merely suspected of rabies. In spite of protective inoculations by, respectively, Pasteur, Ullmann, and Metschnikoff, a certain number of the patients die. Also in the Berlin Institute for Infectious Diseases, 3 out of 853 inoculated persons died of rabies, that is, ⅓ per cent. If the wound from the bite of the inoculated person had been cauterised or subjected to some other antiseptic treatment, which is generally the case, the low mortality can be explained in this way.

The protective inoculation of Pasteur is of no real importance in veterinary surgery, and even in France is looked upon as impracticable for dogs (Nocard). The Government provisions against rabies as enforced in Germany, the regulations to prevent contagion, the dog-tax, and the compulsory use of muzzles in the large towns are fully sufficient for combating hydrophobia.

More recent Methods of Protective and Curative Inoculation.—1. Högyes uses inoculation material in different strengths. He prepares these concentrations from the spinal cord of rabid rabbits with a solution of salt (1 to 5,000, 1 to 2,000, 1 to 500, 1 to 200, 1 to 100, and 1 to 10) and makes successive inoculations, beginning with the weakest concentration. By this method dogs can be rendered immune against both the natural virus of rabies and the fixed virus. Högyes' experiments seem to prove that immunity may be conferred by diluted fresh virus repeatedly inoculated a short time either before or after the animal has been bitten. In 13 cases of dogs which were inoculated subsequent to infection, recovery took place after pronounced symptoms of rabies had appeared. In man, however, recovery from rabies by means of such inoculations has never taken place after the appearance of the symptoms.

2. Helmann and Ferran y Clua have shown that the greater the quantity of Pasteur's inoculation material (brain emulsion) that is injected, the more rapidly and the more certainly is immunity obtained; 8 to 16 fluid drams of the emulsion have, for instance, been injected sub-

* The large majority of English doctors and veterinary surgeons are strongly of opinion that the application of Pasteur's method of protective inoculation to persons bitten by rabid animals, greatly diminishes the risk of the disease becoming developed.—Tr.

cutaneously in one dose. The tolerance of the body to such large doses depends on the fact that the virus is injected only under the skin. Should other parts, such as muscles and nerves, be injured at the same time, the inoculation may be followed by an attack of rabies. The fact which has also been asserted by Helmann, that dogs are also immunised by a single intraperitoneal injection of fixed virus, has been confirmed by Marx.

3. Babes states that Pasteur's method, though effective for obtaining immunity after the bite of rabid dogs, does not succeed in the case of bites by rabid wolves, for which he uses the blood of immunised dogs, and injects $\frac{1}{3}$ oz. of it for each dose in the abdominal region of the bitten person. This inoculation of blood is stated to have been successful in 24 cases. Babes claims, also, to have cured rabid dogs by subcutaneous injections of normal nerve or spinal marrow substance (not confirmed by Calabrese, Anjesky, Gratia and Liénaux). Further, that the internal administration of the liver of rabid dogs confers no immunity (a popular belief) has been experimentally shown by Marx.

4. Galtier has demonstrated that herbivora and omnivora may be rendered immune by intravenous injections, not only against the consequences of subsequent bites, but also against bites already received.

5. Protopopoff placed pieces of virulent spinal cord for, respectively, 30, 46, and 60 days in glycerine broth, and, having thus deprived it of its virulence, he found that one or two intravenous or subcutaneous inoculations conferred immunity against the infection of rabies, and also prevented the appearance of the disease after infection. Out of 19 dogs which had thus been protected, 10 remained healthy after having been infected with the strongest rabic virus, even after it was injected into the brain. Protopopoff actually succeeded, by his method, in preventing an attack of rabies after intracranial infection.

6. Calabrese found that the intraperitoneal serum of immunised rabbits possesses an immunising action against the general virus and the fixed virus. The serum of dogs and of sheep has little or no effect.

B.—RABIES OF THE INDIVIDUAL DOMESTIC ANIMALS

(1.) *Canine Rabies.*

Symptoms.—The clinical appearance of rabies in dogs, as in other domestic animals, occurs in two forms, namely, furious madness, and dumb madness; the former being the more frequent. Pasteur considers that furious madness takes place when the brain is attacked, and also when the virus has been directly inoculated into the brain; and that dumb madness is manifested when the spinal cord is specially invaded, and when the inoculation material has been applied subcutaneously. The two are consequently only different forms of one and the same disease, and are not different diseases, as was held in former times. A furiously rabid dog can transmit dumb madness, and *vice versa*. We must also lay stress upon the

fact that there are many intermediate forms between these two varieties, which often merge into each other so intimately that a distinction is impossible.

Rabies runs a highly typical, acute, and absolutely fatal course.

1. *Furious rabies* has three stages, which are, clinically, in some cases very sharply defined, and in others but slightly :

(*a*) The premonitory or melancholy stage.
(*b*) The irritative or maniacal stage.
(*c*) The paralytic or final stage.

(*a*) The *premonitory stage* of rabies lasts on an average from 12 to 48 hours; and sometimes even longer. The preliminary signs consist especially in the altered behaviour of the animal, which becomes capricious, sullen, peevish, excited, nervous, irritable, frightened, restless, distrustful, or obstinate.* It likes to hide itself, frequently changes its resting-place, and often starts up suddenly ; or it may become extraordinarily affectionate and confiding. Some dogs suffer from an abnormal itching of the site of the bite, which they gnaw and lick. There is a characteristic perversion of taste which has much similarity with that in pica ("licking disease") of cattle. Although at the commencement of the disease the patient has a good and sometimes a voracious appetite, later on it turns away from its accustomed and even favourite food. The affected dog is fond of licking cold objects ; bites and gnaws at whatever comes across its way, and sometimes swallows foreign bodies, especially straw, grass, earth, stones, pieces of wood, bits of glass, rags, and even its own fæces and urine. Some dogs continually smell or lick their sexual organs or those of other dogs. In this stage we may sometimes notice slight spasms in swallowing, inclination to vomit, symptoms of choking, vomiting, coughing, panting, difficulty in breathing, fever, and continued stretching out of the neck. The patient generally suffers from constipation ; in rare cases, from tenesmus. Sometimes there is observed a deviation in the presentation of the disease ; at the outset there may be a complete loss of appetite, while the activity and sprightliness remain normal (Müller).

(*b*) The *stage of irritation* lasts 3 or 4 days, and is characterised by attacks of fury, which may continue for some hours, and convulsions with remissions. Among other signs

* The altered behaviour often takes the form of increased vivacity and increased friendliness of disposition. This high-strung, demonstrative state lasts only about a day or so, and then rapidly changes into the melancholy stage.—Tr.

of restlessness, the dog evinces a violent desire to run away from its home. With this object, very quiet and domesticated dogs, which have been kept in confinement, destroy their kennel or break their chain ; and dogs that live in the house remain close to the door so as to escape ; and when they get into the open they wander about aimlessly, run to and fro, depart on long journeys, or boldly enter strange houses or yards. They often cover in a very short time great distances, 30 or 40 miles a day for instance. In some cases they return home. At the same time they are seized by a more or less excessive morbid desire to bite, which may increase to senseless fury and true mania. At first this inclination to bite is only slight, and is evinced by the dog snapping at inanimate objects, animals or people, and also, in its state of mental confusion, at imaginary objects in the air (snapping at flies). It is also very irritable. Later on it bites, sometimes blindly, sometimes aggressively, everything that comes in its way, such as human beings, including its own master (especially if he provokes it by punishment), horses, sheep, goats, fowls, and particularly dogs and cows. It will even bite iron bars that are held out to it, and live coals. Under such circumstances it frequently bites with such force that it breaks its teeth, or is unable to unlock its jaws. Some dogs direct their fury against themselves, and lacerate their tails, genitals, and legs even to the bone. In rarer cases they carefully avoid man and beast. In a few exceptional instances they show no desire to bite human beings. In this stage we must attach great diagnostic importance to the alteration of the voice, which manifests itself as a peculiarly hoarse, rough, howling bark, the first notes of which are prolonged into a high-pitched, long-drawn-out howl. Paralysis of the vocal chords is the probable cause of this change in the voice.

In other canine patients, depression is more prominent than maniacal excitement. They appear dull and imbecile, have hallucinations, stare constantly at one spot, snap at imaginary flies, howl continually, and are perfectly insensible to blows and kicks. In a few cases well-trained dogs obey the commands of their master to the very last.

(*c*) In the *paralytic or final stage* the animals are usually very much emaciated, frequently to skeletons ; they become disfigured, and often quite unrecognisable ; the hair stands on end ; the eyes are deeply sunken, staring and glassy ; and the appearance is loathsome and weird. At this stage, various

forms of paralysis appear; first of all, that of deglutition, in consequence of which the animal can neither swallow nor excrete saliva. Next comes paralysis of the lower jaw, which drops down; the mouth remains open, and the tongue hangs out. This condition is followed by paralysis of the hind-quarters, which manifests itself in staggering, stumbling, and by increasing bodily weakness. The muscles of the tail, rectum, and urinary bladder become paralysed, with symptoms of weakness and depression, which are varied by paroxysms of excitement that gradually become rarer and rarer. Finally the animal dies between the fifth and eighth, or, as a maximum, on the tenth day of the disease, from paralysis of the brain and general exhaustion. Paralysis of the hind-quarters sometimes appears in the early stage of the disease, before the morbid desire to bite sets in (Zschokke).

Only a few observations on the temperature during rabies have been made. According to Hertwig, the temperature rises about 6° F. or more, and falls rapidly towards the end—a statement which we can confirm from our own experience in one case. Müller found in 2 cases a temperature of 104° F. 24 hours before death.

2. *Dumb madness* is distinguished from the furious form chiefly by the absence, or very short duration, of the stage of irritation or mania. Consequently paralysis, especially of the lower jaw, appears comparatively early. Death takes place in 2 or 3 days. In America, the dumb madness is more frequent than in the raging form.

Several observers state that they have seen recovery after the first stages of rabies. Such instances are certainly extremely rare.

The symptoms of rabies in man are not quite the same as those in dogs, although the disease in both runs through the three typical stages which we have described. In the premonitory stage there is general malaise, pains in the cicatrised wound which was caused by the bite, swelling of the neighbouring lymph glands, and aversion from fluids. In the second stage, reflex spasms, delirium, and mania appear. The reflex spasms affect the muscles of deglutition, and those of the pharynx, and become excited particularly by the sight of water,, by attempts at swallowing (*hydrophobia*), by the feeling of thirst, and even by the mere thought of drinking. These reflex spasms also affect the muscles of respiration and those of the general system. At the same time an excessive feeling of anxiety and uneasiness is present. Thirst is greatly increased, and the patient slavers on account of not being able to swallow the saliva. The temperature of the body is sometimes only slightly increased, but at other times it is considerably raised.

The third or paralytic stage is distinguished by paralysis and spasms, and death takes place in from 2 to 4 days. It has been stated that a few recoveries have been observed. Treatment, apart from prophylactic cauterisation of the wound, consists in bringing on a general narcosis by chloroform, chloral hydrate, potassium bromide, and by the tentative administration of curare. Excision of the wound and cauterisation with nitrate of silver are useless. On the other hand, a deep burning out with the hot iron or corrosion with fuming nitric acid is to be recommended. As disinfecting agents, creolin and corrosive sublimate deserve special mention.

We may see from the foregoing description of the course of rabies in dogs, that the views current among laymen with respect to the symptoms of this disease in these animals are very erroneous. According to popular opinion, rabid dogs always run straight ahead with their tail between their legs; they show great aversion from water; their eyes are excessively red; saliva and froth flow from their mouth, etc. The fact, however, is that rabid dogs not only have no dread of water, but they frequently lap it, and will even swim in rivers and lakes.

Diagnosis.—Positive proof of a bite having been inflicted is of primary importance in the diagnosis of rabies in dogs. The chief *clinical* symptoms are: changed behaviour, altered voice, desire to run away, aggressive and morbid inclination to bite, paralysis, especially that of the inferior maxilla, and the typical course of the disease. If really rabid dogs are segregated and watched, the symptoms just mentioned will become apparent in a few days. On the other hand, the appearance of sugar in the urine should not be credited with a diagnostic importance, as it is not a constant symptom of rabies. If to this we add the negative result of the *post-mortem* examination, we shall be able to make a pretty certain diagnosis of rabies. The presence of foreign bodies in the stomach is, in conjunction with other suspicious circumstances, a valuable diagnostic factor; but it is not sufficient of itself to positively prove rabies, in which disease it sometimes happens that the stomach contains no foreign bodies. Besides, foreign bodies may be found in the stomachs of non-rabid dogs. Sometimes the knowledge of the place from whence the suspected dog came is of diagnostic value. We have, for instance, diagnosed rabies in a dog by the fact that the animal had been recently brought to Berlin from Russia, which is a highly-infected country.

The difficulty of forming a correct opinion is greater in dumb madness, in cases of well-trained dogs being affected, and when the medical history is incomplete, than under opposite conditions. In the former cases, diagnosis can be made only by the intracranial, intramuscular, or intraocular transference of a

fresh piece of brain (pons Varolii, medulla oblongata) to a rabbit. This method is especially necessary in cases in which human beings have been bitten. On this account a regulation exists in Prussia that the official veterinary surgeon is required to transmit the head of the dog suspected of rabies to the Institute for Infectious Diseases in Berlin (in Saxony, the destination is the Veterinary College in Dresden; in Austria-Hungary, the Veterinary College in Vienna, Buda-Pesth and Lemberg respectively). With regard to the methods of diagnostic rabies-inoculation, the following items must be noted:

1. Pasteur introduced the intracranial or subdural method. Rabbits are trepanned with a small instrument; then the inoculation matter is injected beneath the dura mater with a Pravaz syringe furnished with a needle bent at a right angle, and the wound is then sewn up. The inoculated animals die, on an average after from 14 to 20 days, from the dumb paralytic form of rabies (emaciation, paralysis of the hinder extremities, general paralysis). Intracerebral injection is recommended by Leclainche instead of the intracranial (direct injection into the brain substance, after a preliminary perforation of the calvaria by means of a perforating drill).

2. Instead of intracranial inoculation, which demands great attention to detail, Nocard recommends that after depriving the cornea of sensibility with cocaine, the anterior chamber of the eye of a healthy dog should be injected by means of a Pravaz syringe, with a piece of the medulla oblongata rubbed down with distilled water. We may perform the inoculation with greater precision by carefully rubbing down with a little distilled water a small portion of the bulbus of the medulla oblongata, straining the fluid through fine linen, and taking up the filtrate in a Pravaz syringe. Ten to 15 drops of a 5 per cent. solution of cocaine are dropped into the space between the lower eyelid and eyeball of a dog, which is firmly secured. When the cornea has lost its sensibility, it is perforated with the needle of the syringe and 1 or 2 drops of the emulsion are injected into the anterior chamber of the eye.

If the inoculation has been carried out aseptically, only a slight corneal opacity appears at the seat of inoculation. The inoculated rabbits sicken, after an incubation stage of from 12 to 14 days, with the dumb paralytic form of rabies (weakness, paralytic phenomena, emaciation, coma); and usually die after an illness of 48 hours. According to Nocard's experience in over 200 cases, rabies appeared in inoculated animals

after an interval of from 14 to 17 days; Johne found in 22 cases, after from 12 to 23 days (*i.e.*, an average of 18).

3. Beck prefers intramuscular inoculation to the intraocular; as the symptoms of the disease can be traced throughout their whole course in the former, while in the intraocular cases the animals die rapidly, and without distinct paralytic phenomena. In the Berlin Institute, 3 rabbits were as a rule inoculated simultaneously (2 by the subdural method and 1 by the intramuscular, in the long muscles of the back). As in the nervous form of dog distemper, the inoculated rabbits displayed paralytic phenomena. The paralysis did not, however, as in rabies, restrict itself to the hinder extremities, but also attacked the bladder and rectum. Further inoculations of healthy rabbits remain without result in distemper, contrary to that which occurs in rabies.

4. The subconjunctival injection in rabbits is recommended by von Szpilmann as the simplest. Other methods are the subcutaneous; lumbar (between two lumbar vertebræ); intracerebral (through the foramen opticum); and intranasal.

Differential Diagnosis.—There are many diseases which may be confused with rabies, such as hyperæmia of the brain, inflammation of the brain, parasites and tumours in the brain, distemper, epilepsy, inflammation of the stomach, intestinal inflammation, intestinal parasites (*tænia echinococcus, spiroptera sanguinolenta, distomum heterophyes*), foreign bodies in the stomach and intestines, rupture of the stomach (Poisson), perforation of the intestinal canal by *tænia serrata* (Cadéac, Wolpert, and Lahogue), parasites in the nasal cavities (*Pentastomum tænioides*), pharyngitis, bones wedged between the teeth (Johne), foreign bodies and parasites (*tænia, spiroptera sanguinolenta*) in the pharynx and œsophagus, paralysis of the maxilla in consequence of an affection of the *nervus trigeminus* (Vermast, Münich, and Cadéac), facial paralysis, hemiplegia (Sewell), great excitement caused by removal of the puppies from the bitch (Colin, Freer), sunstroke (Fünfstück), love of biting, bad temper, wasp stings, extra-uterine pregnancy (Barzoff), *eustrongylus gigas* in the kidneys, plerocercoides Bailleti in the thoracic cavity, and in the abdominal cavity (Hobday). The fact that the forms of disease named, especially those due to parasites (*pentastomum, tænia, spiroptera, eustrongylus*), are sometimes accidentally found in dogs actually suffer-

ing from rabies, must be borne in mind. In all doubtful cases inoculation is the best test.

Transport of Material Suspected of Rabies Infection.—The regulation which had been established in Prussia requiring the heads of animals suspected of rabies by the official veterinary surgeon to be sent to the Institute for Infectious Diseases in Berlin, has not proved satisfactory, on account of the early occurrence of decomposition of the constituent parts of the brain. It has, accordingly, been recommended to send a small portion of the medulla oblongata to the Institute in sterile water; or, better still, immersed in a flask of glycerine (Frantzius, Kempner).

(2.) *Bovine Rabies.*

Etiology.—Of all the domestic animals, next to dogs, cattle are most liable to become affected with rabies.* Not only are cattle more numerous than horses; but dogs are more aggressive to the former than to the latter. Rabies in ruminants is usually caused by the bite of dogs, and seldom by one ruminant biting another ruminant, as observed by Cope and Horsley among the red-deer in Richmond Park; or by rabid horses, cats, or pigs. The site of the bite is generally on the fore part of the head, especially on the lips, and on the hind-quarters. The period of incubation averages from 4 to 8 weeks, and rarely extends to several months. Mieckley proved in one case that it lasted for 327 days; and Szabo, in another, for 323 days; Morro records 1 of 2 years and 4 months. The minimum duration is 1 week. Spinola states that pregnancy in cows prolongs the period of incubation. As already mentioned, 2 cases of supposed hereditary transmission of rabies to the calf have been published.

Symptoms.—The furious form of rabies is more frequent in cattle than dumb madness. The characteristic symptoms generally manifest themselves by butting with the horns and stamping with the feet. The attacks are often so violent that the horns become broken. Other affected beasts dig up the ground with their horns and feet. The morbid desire to bite is less marked than in dogs. The expression of the face is generally wild; the eyes staring and prominent; and the conjunctiva

* During the years 1887 to 1896, inclusive, the following were the respective numbers of animals which were reported to have been attacked by this disease in Great Britain: 2,386 dogs, 259 deer, 78 sheep, 43 cattle, 21 horses, and 20 swine. The high position in this list occupied by deer is due to the fact that 257 deer were attacked in one year (1887).—TR.

congested. Sometimes the animal continually bellows with a peculiar alteration of the voice. Excessive secretion and dribbling of saliva, and continuous constipation with severe straining to defecate, are constant and striking symptoms of rabies in cattle. This tenesmus may be the first visible sign of the disease. Some patients yawn continually for hours, or display symptoms of meteorism and colic. In many the sexual desire is increased. Rellier indicated as first sign of rabies in cattle a peculiar puckering of the muzzle and upper lip, which gave a curious expression to the face. Other cattle show symptoms of pruritus; that is to say, an extremely violent itchiness of the skin (Martin, Biot). At a later stage the movements of the hind-quarters may become stiff and straddling, and general paralysis may set in. There is finally excessive emaciation, and death takes place in from 4 to 6 days. Ladague has observed, in a few cases, an intermittent course of rabies in cattle.

Differential Diagnosis in Cattle.—We have here to consider: inflammation of the brain, especially tubercular basilar meningitis; poisoning, particularly lead-poisoning; gastro-intestinal inflammation (Papa); foreign bodies in the œsophagus; new growths on the reticulum (Meyer); dermanyssus mites in the outer auditory passage (Stadler and Schuemacher); malignant catarrhal fever; anthrax; rinderpest; sunstroke; excitement in consequence of deprivation of liberty (Köhne); etc.

(3.) *Equine Rabies.*

Etiology.—Rabies in horses is nearly always transmitted by the bite of rabid dogs; in rare instances, by that of wolves or foxes. The period of incubation generally lasts from 4 to 8 weeks, with a maximum of 12 weeks, which, however, appears to have been exceeded in a few cases; for Röll observed one of 283 days; Zündel, one of 378 days; Fambach, one of 472 days; while Gotteswinter, Fisch and Swain have noted, respectively, instances of 20, 21 and 25 months.

Symptoms.—Rabid horses at first exhibit unsteadiness, restlessness, and terror. They paw the ground with their feet, neigh, gnaw and bite at the manger, partitions and articles of stable gear; and sometimes manifest an itching irritation on

the site of the bite, which is mostly found on the lips, muzzle, or forelegs. Some, but by no means all, rabid horses are aggressive to mankind and become seized with an irresistible impulse to bite and kick. Some lacerate and mutilate their own bodies by tearing the skin, testicles, etc. Others will rush like a dog at a stick if it is held out to them, and will bite and pull at it. By the violence with which they bite hard objects, they sometimes break their incisor teeth, and even the body of the inferior maxilla. Certain horses remain in a dazed condition; and though at first they eat ravenously, they will not touch their food later on. Other horses masticate everything that gets between their teeth: they even take up their dung and swallow it, and evince severe thirst. Great desire to micturate and increased sexual excitement are characteristic of equine rabies. Stallions try to mount mares, have erections, and involuntarily eject semen. Mares become violently in season, scream, strain to urinate, and manifest excessive irritation of the pudenda. When paralysis of the muscles of deglutition sets in, the food is returned through the nose; the animal works its jaws continually, as in chewing, grinds its teeth, shakes its head, and swishes its tail. These symptoms are associated with convulsions and muscular spasms in different parts of the body, especially in the muscles of the head (lips and cheeks), neck, abdomen, and thorax. Symptoms of colic, such as groaning, frequent lying down and getting up, straining to defecate, and even prolapse of the rectum may occur. Finally, general paralysis sets in, the animal staggers in its walk, knuckles over, gives way at the fetlocks, and exhibits a groping, uncertain gait, and, as a rule, soon becomes paralysed in the loins. In some cases, paralysis appears at first in the muscles adjoining the wound, as on the lips or on a forefoot (Gerlach and Mégnin). The temperature of the body rises from normal to 104° F. or more; and the beats of the pulse may be increased to double or treble their usual number. Towards the end there is frequently a general outbreak of sweat. Death often takes place by apoplexy as early as 24 hours. The average duration of the disease is from 4 to 6 days. Dumb madness, in which the morbid desire to bite is absent, is more common in horses than in cattle.

Differential Diagnosis.—In horses the following diseases may be mistaken for rabies: Inflammation of the brain, staggers, abscesses of the brain (Kopp), excessive horsing, colic, inflammation of the stomach (Olivero and Allemanno), rupture of the

stomach, and paralysis of the loins. The psychical disturbances, morbid desire to gnaw and bite, straining to defecate and micturate, symptoms of colic, local itching irritation, and increased sexual desire are phenomena of diagnostic importance.

Protective Inoculation.—Nocard and Leclainche have recommended protective inoculation against rabies for horses in the form of an intravenous injection of the filtered emulsion of a medulla oblongata taken from a rabid animal; Conte has found the procedure effective.

(4.) *Rabies in Cats.*

Symptoms.—After an average period of incubation of from 2 to 4 weeks, rabid cats show great inclination to run away from home, and a strong desire to bite and scratch. The injuries caused by rabid cats are more dangerous than those inflicted by any other domestic animals; because cats jump up on persons, and try to lacerate and scratch the face. They also attack dogs, of which they show no fear. Their voice becomes characteristically altered and hoarse. Death takes place between the second and fourth day. Rabies of cats may be confused with helminthiasis. Next to dogs, cats are the most frequent agents by which rabies is transmitted to man. Pasteur estimated that among 12,000 pe sons who had been bitten by rabid animals, 11,000 were bitten by dogs, and more than 700 by cats.

(5.) *Rabies in Pigs.*

Symptoms.—Rabid pigs show at first great restlessness, run wildly about, scream, squeak, dig up their litter, hide themselves, attack man and beast, and exhibit a great desire to bite. They secrete an abnormally large amount of saliva, slaver, and frequently swallow foreign bodies. At the commencement of the outbreak, the infection wounds often manifest a well-marked inflammation, which induces the animals to rub and gnaw the wounds. It has been stated that dumb rabies has not been observed in pigs. The duration of the disease is very short, and usually lasts only 1 or 2 days; frequently only 1 day. The period of incubation is on an average 2 or 3 weeks. According to the Prussian statistics of epizoötics, it varies from 6 to 179 days.

(6) *Rabies of Sheep and Goats.*

Symptoms.—Rabid *sheep* show great restlessness, and considerable pruritus at the site of the bite, which they lick and gnaw, and pull out the wool near the part. Sexual desire is usually considerably increased. The affected animals crowd and push against their fellows, try to jump on them, bleat with a dull and rough voice, and lose all shyness and timidity towards mankind. If people approach them they become very restless, stamp with their feet, bite at a stick if it is held in front of them, and will bite even stone. They try to jump up on walls, snort wildly, and assume a glaring and staring expression of face. Excessive weakness with paralysis of the hind-quarters soon sets in, and the sufferers generally die in from 3 to 6 days. The period of incubation is on an average 3 or 4 weeks. In most cases, the infection takes place through dogs, by which a great part of an entire herd may become rabid. In rare cases the infection is due to a rabid wolf or bull (Bourrel).

The symptoms in *goats* are essentially the same as in sheep. Galtier observed restlessness, great agitation, wanderings of the mind, plaintive bleating, aggressive advances towards men, distracted lickings, butting and biting of the kids, and death after 8 days.

(7.) *Rabies of Birds.*

Symptoms.—Rabid birds are very easily frightened. They become extremely restless, run about continually, screech, and jump about in a disordered manner. They attack each other and even human beings with beak and claws, and may thus tear pieces of cloth from people's clothes and try to swallow these bits of cloth. Their voice soon becomes hoarse; they stagger; become paralysed; and die in 2 or 3 days. The period of incubation is about 6 weeks, with a maximum, it is stated, of 11 months.

According to the experiments of Kraus and Clairmont (subdural inoculation), fowls, geese, ducks and young pigeons are made rabid by the waste virus as well as by the fixed. Older pigeons appear to be immune, while younger pigeons and geese die with symptoms of ataxy, paresis and paralysis. Fowls sicken first after from 1 to 2 months with ataxic-paretic symptoms; then apparently recover; and finally succumb with paralysis of the cervical muscles. Older pigeons may be made susceptible to the infection of rabies by having food withheld from them.

FOOT AND MOUTH DISEASE.

Foot and mouth disease in cattle—Foot and mouth disease in sheep—Foot and mouth disease in pigs—Infectious aphthæ of horses—Infectious aphthæ of dogs—Infectious aphthæ of cats and rabbits—Infectious aphthæ of birds—Transmission of mouth disease from cattle to man.

History.—Foot and mouth disease (*aphthæ epizooticæ*) has been known for a very long time. It was well described by some observers in the middle of the eighteenth century, when it broke out with great violence, especially in Germany and France. It raged from 1809 to 1812 and from 1819 to 1823, particularly in Southern Germany, Switzerland, and Italy (Buniva, Handel, Lux, and others). In 1839 it was introduced into England, where, since then, ten great outbreaks have taken place (Brown). In the forties (1845–1846) and fifties (1855–1857) it spread over the whole of Europe. Among recent epizootics we may mention those which occurred in the years 1862, 1869, 1871–1874, 1875–1877, 1883–1884, 1892, 1896, and 1897. During 1871, 700,000 head of cattle, both in England and France, became infected, with a loss of 7,000 head (1 per cent.); during 1872 in Würtemberg alone, 50,000 head, with a mortality of 1,500 (3 per cent.); and during the same period in Baden, 150,000 head of cattle were attacked. In 1883 in Great Britain, the disease attacked nearly half a million; in Prussia, Austria and Italy, 60,000 in each country; and in Bavaria, 100,000; in the German empire during 1892, over 4,000,000 (in Prussia alone over 3,000,000); in the years 1896–1897, taken together, nearly 3,000,000 animals died of foot and mouth disease. The last great epizootic invasion of foot and mouth disease in Germany took place during the years 1890 to 1892. In some years this usually benign disease has assumed a very malignant type, as many cases of death or of serious sequelæ have resulted, particularly in 1892, when foot and mouth disease appeared in a very destructive form. For instance, the loss in Bavaria alone during the months of August, September and October of that year was over 3,000 head of cattle. In Würtemberg, 1,500 cattle died in the year 1896; 1,300 in 1898; as well as numerous sheep and pigs, of a malignant type of foot and mouth disease. Similar experiences had previously occurred. Thus 2,000 head of cattle died from foot and mouth disease during 1839 in the cantons of Berne and Freiburg. In 1872 there were heavy losses from it in France and Bavaria.

The distribution of the disease as an epizoötic follows the routes of traffic. The observation has often been made that its course was directed from east to west, so that it has frequently extended over wide areas in a very short time; for example, from the Caspian Sea to the Atlantic Ocean. The more extensive the network of railways is in a country, the more rapidly does the plague spread.

Etiology.—The agent of infection in foot and mouth disease is not yet known. All the researches which have hitherto been made for the purpose of demonstrating it by staining, and of reproducing it by cultures, have given negative results. The important researches made with such thoroughness at the Prussian Institute for Infectious Diseases in Berlin; in the Hygienic Institute at Greifswald; and in the German Sanitary Office (Plague Commission, 1897–1903), have remained without result. This Commission has definitely demonstrated that the causative factor in foot and mouth disease is distinguished from nearly all other known micro-organisms by the fact that it passes through the usual bacteria filters. This peculiar property, and that of non-staining, are apparently to be attributed to its being of much smaller size than the micro-organisms which have hitherto been discovered.

With regard to the distribution of the infective material in the body of the affected animal, the mode of infection, and the tenacity of the contagium, we have, on the other hand, numerous older experiments of veterinary practice, as well as the more recent researches of the Plague Commission above referred to.

The causative agent of foot and mouth disease is chiefly present in the contents of the vesicles on the mucous membrane of the mouth, on the foot, the udder, and the vaginal mucous membrane. It is also found in the secretions and excretions, which are infected with the lymph of the vesicles (saliva, milk, dung and urine). It appears to be present in the blood only during the height of the fever, and even then but in relatively small quantities. Experiments on feeding with frozen flesh, liver, spleen, kidneys, etc., have hitherto proved entirely negative. Distribution of the infective material by way of the atmosphere, according to recent researches, is not possible.

The infective agent of foot and mouth disease is very susceptible to disinfecting agents, to high temperature and to drying. Its virulence becomes extinct after 24 hours' drying at ordinary chamber temperature.

By boiling (212° F.), the fluid of the vesicles becomes at once inert; the same effect is obtained by heating for from 10 to 30 minutes at 140°–158° F. Heating to 185° F. destroys the infecting power of milk to which the lymph has been added. In manure heaps, also, the virus of foot and mouth disease does not long continue infective, at a depth of over a foot, when their temperature, in consequence of decomposition, rises to 158° F. (Hecker). On the other hand, the virus is very resistant to the influence of low temperatures; in ice-chests the lymph retains its activity for a month; nor will several hours' lowering of the temperature of the lymph of the vesicles (—54.4° F.) deprive it of its virulence. Even quite feeble disinfecting agents (such as milk of lime, a 3 per cent. solution of soda, 1 per cent. solution of carbolic acid) destroy the contagium by one hour's exposure. Finally, while the infecting material is well preserved in fresh milk, it rapidly perishes in sour milk, or that to which rennet has been added.

The digestive apparatus forms the chief entrance for the contagium. This is confirmed by the well-known fact that the disease can be transmitted, if we rub the lymph of the vesicles on the previously injured mucous membrane of the mouth of healthy cattle (inoculation). Infection can also take place through the stomach and intestine (experiments with lymph enclosed in gelatine capsules). Other ports of entrance are: the nasal mucous membrane, air-tubes, conjunctiva of the eyelids, the teats, and the anus. Experimentally, the disease is most easily produced by intravenous and intraperitoneal injection of the lymph of the vesicles; on the other hand, subcutaneous injection leads to no result.

The transmission of foot and mouth disease from sick to healthy animals is either direct or indirect. Direct infection is especially carried out by mutual lickings of animals; and, in the case of sucklings, from the udder or through the milk. Intra-uterine infection has also been often observed (aphthæ and ulcers of the mucous membrane of the mouth in new-born calves). The indirect form of infection is often caused by means of numerous media, such as skimmed milk and waste products of dairies; infected dung; common pastures and drinking troughs; cattle markets; cattle-yards; stalls and inns; flocks of sheep and droves of pigs; animals affected with the disease; cattle-dealers; drovers; servants; dogs; cats; and fowls (see Vol. II., p. 594).

Bacteriology.—By means of the researches in the Institute fo Infectious Diseases and in the Sanitation Office, it has been establishe

that no agent of infection known in literature stands in causal relationship with foot and mouth disease. Nosotti states that he has constantly found in the pure contents of the vesicles, a micrococcus which can be easily stained in a solution of aniline. He made pure cultures of it in nutrient material outside the body, and inoculated it successfully. Klein claims that the infective matter of foot and mouth disease is a micrococcus which he cultivated artificially, and which produces characteristic cultures as a diplococcus and streptococcus, 20 or more cocci being sometimes attached to one another. Klein made these cultures on suitable nutrient material (solid blood serum and solid nutrient gelatine), and found that they assumed the appearance of soft, punctiform, granulated membranes, which consisted of small drops lying close together. He states that he produced foot and mouth disease in sheep by feeding experiments with these cultures, but that subcutaneous inoculations gave negative results. Schottelius states that the cause of infection is spherical formations which are attached to each other in the form of a rosary, and which resemble streptococci, and consequently he has given them the name of *streptocytes*. Subcutaneous injections of pure cultivations produced in cattle a feverish affection accompanied with salivation. Kurth found a similar organism and called it *streptococcus involutus*, the cultures of which gave negative results by inoculation. Special micro-organisms have been discovered also by: Bussenius and Siegel, Rivolta, Sanfelice, van Nissen, Starcovici, Stutzer, Babes and Proca, Piana, and Fiorentini, Jungers, Behla, and others.

Occurrence.—Foot and mouth disease is a malady peculiar to ungulates, and therefore occurs chiefly in cattle, sheep, pigs and goats. Horses, dogs, cats, and birds are less frequently attacked. Human beings become easily infected. All wild even-toed animals (red-deer, roebucks, fallow-deer and chamois) and wild ruminants in zoological gardens (camels, llamas, giraffes, antelopes, aurochs, buffaloes, yaks, zebus,* etc.), and also elephants, are liable to it. Every kind of animal can infect the others: for example, cattle and sheep are infected by pigs; sheep by cattle, and *vice versâ*. The disease is also sometimes communicated by affected deer.

Foot and mouth disease is of great importance from an agricultural point of view; for the pecuniary loss is generally very severe, in consequence of the rapid manner in which this disease spreads through the entire stock of a country. From 25 to 50 per cent. of all the cattle may become infected; traffic and commerce suffer great restrictions in consequence of the legal enactments that are put into force; the loss in milk and its products is heavy; sometimes the mortality of the infected animals is comparatively high; and the value of the survivors is depreciated by decrease in their working powers, and by diminution

* The zebu is not found in a wild state.—Tr.

in their bodily weight. The losses in Germany in 1892 were estimated at over £5,000,000 (4,000,000 of affected animals; of which 1,500,000 were cattle; the total loss from the latter, in milk, loss of condition, and deaths, was calculated at £2 10s. per head). In the province of Saxony, the total loss in 1899 was estimated at £1,000,000 (£5 7s. per head). The losses thus caused in 1883 in England were estimated at a million sterling; in France in 1871, at £1,500,000.

Statistics.—In Germany, from 1886 to 1888, only 100,000 animals were infected with foot and mouth disease; but in the next 14 years (1889–1902) collectively, over 16,000,000 animals were attacked by the disease. The respective figures are as follows:—

Year.	Total.	Cattle.	Sheep and Goats.	Pigs.
1886 . . .	13,603	5,366	6,308	1,839
1887 . . .	31,868	12,723	14,400	4,745
1888 . . .	82,804	37,134	19,786	25,884
1889 . . .	555,178	262,375	238,399	54,404
1890 . . .	816,911	432,235	230,868	153,808
1891 . . .	821,130	394,640	244,282	182,208
1892 . . .	4,153,537	1,504,299	2,210,986	438,252
1893 . . .	500,342	204,832	220,402	75,108
1894 . . .	192,613	93,921	66,287	32,405
1895 . . .	464,636	195,120	210,950	58,566
1896 . . .	1,548,437	710,481	585,888	252,068
1897 . . .	1,163,870	537,969	449,674	176,227
1898 . . .	852,968	462,078	269,783	121,107
1899 . . .	4,266,001	1,885,774	1,565,365	814,862
1900 . . .	1,046,149	430,356	415,499	200.294
1901 . . .	183,139	80,739	76,022	26,378
1902 . . .	42,459	20,144	15,168	7,147

From these figures we find that the number of affected sheep and goats is about the same as that of cattle. It is remarkable that foot and mouth disease, according to the researches of the Plague Commission of the Sanitation Office, and of the Institute for Infectious Diseases, is not transmissible to sheep and goats by inoculation.

The following factors favour its dissemination: introduction from neighbouring states; neglect or defective execution of the police regulations with regard to the importation of cattle; defective disinfection of sheds and cattle-trucks; unrestrained use of animals for draught purposes; unrestrained use of common pastures and of wells for drinking; cattle dealing; driving cattle along high roads; removal of manure; intermediate bearers, such as human beings (attendants on cattle, butchers, castrators of cattle, servants, postmen, inspectors of churches and of schools, neighbours, relatives, pedlars, milkmen, and sometimes veterinary surgeons and their clothes); cattle food; dung utensils; undressed skins; blood; horses; cats; geese; poultry (even storks, crows, wild ducks, starlings, magpies, rats, mice, flies); and various common articles; un-

sanitary dwellings ; over-flowing of middens ; and dairy companies. The skimmed milk and waste products which are returned by such dairies, and are given to pigs and calves as food, largely contribute to the spread of the disease. Stress is also laid upon the fact that the permitted practice of sending out the unboiled milk from healthy cows which were living in infected sheds was a concealed source of considerable danger. Favourable results in extinguishing the disease, and in restricting its dissemination, were obtained in several places by temporarily prohibiting the holding of cattle-markets, the suppression of which was not, as a rule, followed by any economic disadvantages.

[From 1886 to 1896, inclusive, the number of animals attacked by foot and mouth disease in Great Britain, were as follows : cattle, 1,295 ; sheep, 3,673 ; and swine, 107. No animals were reported to have been attacked during the years 1887, 1888, 1889, 1890, 1891, 1895, 1896 and 1897. Since the year 1877, more sheep appeared to have suffered than cattle.—Tr.]

Incubation Stage.—The time which elapses between the entrance of the contagium of the disease into the body, and the first visible symptoms of the disease (the so-called incubation stage), is, in cattle, on an average from 3 to 6 days. The minimum period of its duration is from 24 to 36 or 48 hours ; the maximum from 7 to 14 days. These figures, which refer to the natural infection, differ so widely because the stage of incubation depends upon various factors. Of these are especially to be mentioned, the quantity and virulence of the infecting material, the method and manner of the transmission (various modes of entrance into the body) ; and also the individual disposition of the different animals (age, sex, bodily constitution, individuality).

The duration of the incubation depends upon whether the infection which takes place is natural or artificial. In artificial communication (inoculation) this reaches, on an average, in cattle only from 48 to 60 hours (24 hours to 10 days)

Finally, the species of animal has its influence on the stage of incubation. Among these, the shortest is that of pigs, in which the natural infection gives an average duration of but from 24 to 48 hours. The average period in sheep (inoculation) lasts from 24 hours to 6 days.

Symptoms in Cattle.—Foot and mouth disease manifests itself in cattle by the appearance of vesicles and ulcers on the oral mucous membrane (mouth disease), and on the skin of the coronet and on the skin of the interdigital space (foot disease). Sheep, goats, and pigs are usually affected only on the feet.

Symptoms of the oral form of the disease in cattle.—After a period of incubation of, on an average, from 3 to 5 days (with a minimum of from 36 to 48 hours), there is a moderate rise of temperature (up to 104° F.), which becomes normal as soon as the eruption appears. Appetite and secretion of milk become diminished; rumination is suppressed; the mouth is generally kept closed; and slight salivation takes place. After 2 or 3 days yellowish white vesicles, the size of a hemp seed, appear on the gums, especially on the toothless part of the anterior maxilla; on the edges, base and lower surface of the tongue; on the buccal mucous membrane; and on the lips. These vesicles gradually increase in size, so that they sometimes become as large as a five-shilling piece or even larger; and they are also often confluent. These vesicles, large and small, stand out prominently from the surface and contain at first a yellowish, limpid fluid, which gradually becomes turbid. The bursting of the vesicles leaves on the affected mucous membranes moist, highly reddened, and very painful erosions, denuded of epithelium. These spots become slowly covered with fresh epithelium or turn into ulcers with a foul base and finally cicatrise. At this stage of the disease there is a considerable amount of salivation and slavering. The saliva continually runs in long threads out of the mouth and accumulates in the feeding-trough. The "smacking" noises thus produced by the movements of the jaws are very characteristic. The exanthema usually spreads to the muzzle. During the course of the disease the animals become greatly emaciated, with a loss of weight which may be as much as 1 cwt. The milk is altered and assumes a mucous, colostrum-like condition, a yellowish-white colour, and has a bad taste. It coagulates easily, and can only with difficulty be turned into butter and cheese. The quantity of the milk becomes considerably decreased, not only during the disease, but also for a comparatively long time after recovery. This reduction varies, on an average, from 50 to 75 per cent.; after the lapse of the disease, also, the yield of milk is diminished in quantity for a considerable period. The exanthema in milch cows often spreads by the act of milking to the udder and teats.

The foregoing symptoms are those of the simple and ordinary form of the disease, which runs its course in from 8 to 14 days. At other times it assumes a *malignant type* which is entirely different from that of the benign form, and has been carefully studied only within the last few years. The most

striking feature of the severe variety of foot and mouth disease, which may have very unusual sequelæ, is a fatal complication resembling apoplexy, leading to death with symptoms of the character of paralysis, at the height of the fever or during the period of convalescence, after the exanthema has healed. The animal begins to bellow and falls dead quite suddenly, as if struck by lightning, or dies in a few hours under symptoms of violent dyspnœa, discharge of blood from the nose, and great prostration. The cause of these apoplectic cases of death, which remind one of anthrax, is paralysis of the heart or paralysis of the lungs, produced by the toxins in the infective matter of the disease, or in a lodgment of food and drink in the upper air-passages, resulting from paralysis of the larynx and pharynx. More rarely we meet with a paralytic weakness of the hind-quarters, resembling the paresis which follows parturition.

There are also peculiar local manifestations of the affection in different parts of the body. The inflammation spreads from the canals of the teats to the parenchyma of the mammary glands and leads to the development of violent inflammation of the udder, followed by sero-sanious discharge from the teats, induration, and atrophy. If the aphthæ appear on the pharyngeal mucous membrane, the animal displays symptoms of pharyngitis, difficulty in swallowing, regurgitation, and cough, and sometimes contracts pneumonia due to foreign bodies, especially when drenches have been administered. Nasal catarrh and bronchial catarrh are often present, as in other acute exanthemata. If the exudative process in the oral cavity be very intense, croupy or caseous plate-like formations may appear upon the oral mucous membrane with disintegration of the cast-off epithelium, in consequence of which a fœtid odour may become developed in the mouth. Some affected animals, especially sucking calves, and also full-grown cattle, show in grave invasions of the disease, an intense gastro-intestinal affection with severe diarrhœa. We may sometimes observe inflammation of the matrix of the horn cores, and vesicles on the skin at the base of the horns, which may even be shed. In a similar way we may find vesicles on the vulva, vaginal mucous membrane, entire skin, especially on the abdomen and chest, and even on the cornea. Abortion is not infrequent during the attack and a short time after recovery. In a few instances the exanthema is entirely absent, in which case the manifestations of the disease somewhat closely resemble those of anthrax fever (subacute anthrax, Vol. II., p. 541).

Symptoms of the pedal form of foot and mouth disease in cattle. —The pedal symptoms generally appear after the oral manifestations, although they may be the first to develop. We observe first of all redness, abnormal heat and painful swelling of the skin at the coronet, and especially between the toes and towards the plantar cushions. These changes may appear on 1 foot or on all 4 feet at the same time. Minute vesicles the size of a pea up to a hazel-nut, spring up after a day or two on the inflamed skin, and soon burst. They are filled at first, as already stated, with a yellowish, limpid fluid, which later on becomes turbid and greasy. At the same time the animal goes lame, moves stiffly and lies down a good deal. Under ordinary circumstances the vesicles heal completely in from 8 to 14 days.

Also in the pedal form, grave complications are not rare. Severe inflammation may appear on the skin of the digits, especially when the disease has been neglected and when the animal has been compelled to stand on damp and soiled ground, or when it has had to travel over rough and stony roads. In such cases, the aphthous process becomes complicated with exciters of septic inflammation which lead, first of all, to an erysipelatous inflammation of the skin of the digits; and later on to the formation of ulcers and abscesses, panaritium in all its forms, purulent arthritis in the neighbouring articulations, necrosis of the bones, shedding of the hoofs, and finally to septicæmia and pyæmia. In such cases we find considerable decubital gangrene.

Anatomical Conditions.—The changes found in malignant foot and mouth disease vary greatly. Death may be due to congestion and œdema of the lungs, and passive dilatation of the heart; to fatty degeneration, hæmorrhagic infarcts and cellular infiltration of the muscular portion of the heart; or to violent gastro-enteritis with intestinal hæmorrhage. Multiple embolic myocarditis has also been found to be present. The *post-mortem* appearances are sometimes very slightly characteristic. We may find in the abomasum a violent inflammatory œdema of the mucous membrane with prominent gelatinous swellings; and in the first 3 stomachs, especially along the pillars of the rumen, aphthæ, ulcers, and hæmorrhagic foci. The mucous membrane of the small intestine is sometimes glassy, swollen, dark in colour, and infiltrated with inflammatory hæmorrhages, and the contents of the intestine bloody. Aphthæ have been found in the larynx and pharynx and on the inflamed tracheal

and bronchial mucous membrane. In a few cases pleuritis, hydrothorax, hydrocephalus internus, cerebral and spinal œdema and subcutaneous hæmorrhages were observed on *post-mortem*. Finally, the aspect of septicæmia has been noticed in many animals.

Malignant Foot and Mouth Disease.—The malignant form of foot and mouth disease of cattle, of which the symptoms and pathological changes have already been substantially described (see above), has been for a long time known to veterinary practitioners. Communications thereon had already been made by Rychner (1841). During the past decade, exhaustive researches have been carried out with the view of ascertaining the nature of the so-called malignant foot and mouth disease; especially in Saxony (Johne and others), Switzerland (Guillebeau), Bavaria (Kitt, Imminger, Mayr, and others), Austria (Csokor, Schindelka), Baden and Würtemberg. Etiologically, the disease still remains so far without elucidation that the proper cause of the malignancy is not known (a specially virulent contagium (?), absorption of paralysing toxins through the digestive mucous membrane which has been altered by inflammation (?)). Bad housing of the animals is not to be accepted; as a malignant course has been observed in even model sheds. The anatomical conditions are not always the same. Death appears to occur most frequently as a result of paralysis of the heart (parenchymatous myocarditis). In other cases we find definite symptoms of suffocation and pulmonary paralysis (foreign bodies in the paralysed larynx and pharynx, congestion of the lungs, œdema of the lungs). Hæmorrhagic inflammation of the small and large intestine and numerous aphthæ and ulcers of the ante-stomach have also been found. The appearances of intoxication (poisoning) have also been observed (hæmorrhages, especially in the heart-substance), also those of septicæmia (parenchymatous swelling of the internal organs). It is a striking fact that the aphthæ, which form the distinctive signs of foot and mouth disease, are frequently found to be already in the process of healing.

The treatment is one directed to symptoms (alcohol, atropin, digitalis, and other cardiac remedies); as regards prophylaxis, Mayr recommends the earliest possible disinfection of the contents of the belly with creolin.

Differential Diagnosis.—We must not confuse the ordinary mild form of the disease with injuries, cauterisations and scalds of the oral mucous membrane; aphthous and mycotic stomatitis produced by fungi in the fodder and known as "sporadic aphthæ;" actinomycosis of the tongue; stomatitis mercurialis; poisoning with potato-tops; variola; panaritium; inflammation of the cleft of the hoof; eczema of the back of the pasterns from feeding on distillers' wash; and ordinary grease (eczema of the pastern). In one case Henninger saw an infection of the oral mucous membrane brought on by licking greasy heels. The malignant variety of the disease is frequently confused with anthrax and sometimes with the bovine form of "deer and cattle disease" and rinderpest.

Symptoms in Sheep.—In sheep and goats the manifestations of the disease are usually confined to the feet. The inflammation attacks the edge of the coronet, and extends to the cleft of the hoof, or, behind, to the plantar cushions. The coronet is swollen, reddened, and exudes a serous fluid. The affected animals go lame and lag behind the herd. We may sometimes find aphthæ on the toothless edge of the upper jaw, upper lip, gums of the inferior maxilla, and pudenda. Among sheep, sucking lambs become most gravely affected, and frequently die in a very short time under symptoms of severe gastric derangement. In full-grown animals the course of the disease is usually acute and benign. The affection runs its course much more slowly in a flock of sheep than in a herd of cattle or in a drove of pigs. The period of incubation is from 2 to 8 days.

The chief complication is the malignant form of foot disease, which appears to be a mixed infection of aphthous inflammation of the hoof and panaritium. Its presence gives rise to symptoms of erysipelas, lymphangitis, cellulitis, serous decomposition of the secreted horn, formation of abscesses and suppurative undermining of the hoof, separation of the horn at the coronet, shedding of the hoof, purulent inflammation of the glands of the hoof, purulent arthritis, necrosis of the bones, and pyæmia. The chief predisposing causes are: moisture, dirt, over-exertion from long journeys, excessive wear of the hoofs from bad roads, injuries from travelling over stubble-fields, etc.

The disease may be mistaken for ordinary traumatic eczema of the skin of the hoof, which, however, is never contagious, shows no aphthæ, and runs a more chronic course. Hess states that he has observed a contagious, malignant inflammation of the hoof which is independent of foot and mouth disease, and is probably caused by the bacillus necrophorus.

Foot and Mouth Disease in Pigs.—Here, also, the disease manifests itself more frequently on the feet than in the mouth. The period of incubation varies, on an average, from 36 hours up to 3 days, with a minimum of 24 hours. The disease appears to be uninfluenced by breed. The changes are similar to those in sheep. The chief symptoms are the vesicles and the inflammation of the coronet, with consequent lameness. We find in pigs the same complications as in the malignant foot disease of sheep, especially among pigs which are driven in herds. In the oral form of the disease, to which sucking-pigs are especially predisposed, the eruption of aphthæ spreads to the snout, tongue and palate, in the form of vesicles the size of a pigeon's egg up to that of a walnut. As causes of death from the malignant form, we may

mention œdema of the lungs, broncho-pneumonia, hydrothorax, and purulent pleuritis. With regard to differential diagnosis, contusions of the plantar cushions from long journeys are of special importance.

Infectious Aphthæ of Horses.—In horses we find only the oral form of the disease. The infection is conveyed by cattle, goats, sheep and pigs. For instance, 1 case was caused by a horse licking a bullock which was suffering from foot and mouth disease (Bräuer). In other instances the disease was transmitted by a water-bucket. Many of the reported cases of foot and mouth disease in the horse should, however, be regarded with great suspicion; for evidently several of them were either those of stomatitis pustulosa contagiosa or stomatitis catarrhalis. Nevertheless, foot and mouth disease has been positively proved to have occurred in horses. The symptoms are the same as in cattle, and consist of fever, loss of appetite, eruption of vesicles on the oral mucous membrane, lips, and even on the nasal mucous membrane and skin, and sometimes catarrh of the stomach, intestines and bronchi. It is said that croupy processes occur more frequently in horses than in cattle. Saïkin saw 27 remounts of a Russian brigade affected with foot and mouth disease; it was not communicated to the older horses; recovery usually took place in from 8 to 9 days; the complications were swelling of the laryngeal glands, nasal catarrh, and throat catarrh.

Infectious Aphthæ of Dogs is a very rare disease; although stomatitis aphthosa and ulcerosa and inflammation of the skin between the toes and of the pads of the feet, have been seen in dogs which had come in contact with infected cattle or pigs, or which had been kept in infected stalls. By inoculation, the disease may be communicated to young dogs also (Hecker).

Infectious Aphthæ of Cats and Rabbits.—Vesicles and ulcers on the mouth and tongue, upper lip, and anterior part of the head, with fever, vomiting, and lameness, have been observed in cats and rabbits. The disease may also be transmitted by inoculation to cats, rabbits and guinea-pigs (Hecker).

Infectious Aphthæ of Birds, which very seldom occurs, manifests itself by vesicles on the tongue, pharynx, nasal mucous membrane, conjunctiva, comb, and feet, including the web between the toes. Experiments by Kitt and Schindelka in the transmission of the disease to birds gave negative results.

Transmission of Mouth Disease from Cattle to Man is by no means rare, and is more frequently seen by veterinary surgeons than by physicians. Children are specially liable to become infected by drinking unboiled milk from cows suffering from foot and mouth disease, and sometimes die from the consequences of the infection. Transmission may take place by butter and cheese, through wounds on the hands and arms, and indirectly by intermediate bearers. The symptoms of the disease in man consist of fever, disturbances of digestion, eruption of vesicles on the face (lips and ears), fingers, arms, chest, mucous membrane of the mouth, pharynx (angina) and conjunctiva, and diarrhœa accompanied by vomiting, which may cause the death of young persons.

It appears that the disease cannot be transmitted by eating the flesh of infected animals. It is possible that the severe skin affections which have appeared in some years (1883–84, for instance) in children after vaccination, were really foot and mouth disease and not cow-pox.

We take the following brief notes on the transmission of foot and mouth disease from animals to men, from the very rich literature which exists on the subject. Sagar states that in 1764 several people became infected in Moravia. In 1778 all the inhabitants of a monastery had an attack of vesicles in the mouth after drinking milk. Berbier (1811) reports similar cases in the department of Rhône in France; and Tilgner (1834) in Germany. Hertwig, Mann, and Villain in 1834 carried out the experiment in their own persons of drinking, for 4 consecutive days, milk warm from a cow which was suffering from a severe attack of foot and mouth disease. All 3, especially Hertwig, became ill on the second day with fever, which was followed by an eruption of vesicles on the mouth, fingers, and hands. Schneider observed, in 1840, in men violent abdominal pains, vomiting, and bloody and copious diarrhœa after drinking the milk of a cow the udder of which was affected by aphthæ. Butter and cheese have also been proved to be dangerous. From eating cheese, a male servant suffered from the oral form of foot and mouth disease and nervous fever, a peasant woman died of malignant mouth disease; and whole families became ill. During an enzoötic of the foot disease in 1845, Gierer saw aphthous affections of the mouth in children. Hulin states that out of 1,000 inhabitants of a village, 23 died of infective aphthæ. Holm saw vesicles on the nipples of a woman who was giving milk to an infant, and Guilmont found in 2 girls aphthæ on the mouth and hands. Zürn reports an instance of death from drinking the unboiled milk of an infected cow, in the case of a child whose gastro-intestinal mucous membrane showed numerous vesicles and ulcers on *post-mortem*. According to reports of Prussian veterinary surgeons in 1874, many people became ill from drinking butter-milk. Demme (1876) saw aphthous stomatitis in twins, 1 of whom subsequently died, and the mother also became affected by the same disease. Stang (1882–84) saw 2 women with aphthæ in the mouth, nasal cavities, space between the fingers, and on the arm. In the district of Breslau (1885) severe cases occurred in children. One child 13 years of age had high fever, violent conjunctivitis, and aphthæ in the mouth; and another 9 months old suffered from diarrhœa and vomiting, and died. Esser saw 4 children affected by vomiting, headache, pains in the limbs, and great weakness. Haarstick, von der Ohe, Bührmann, Eggeling, Noack, Weigel, Walther, Hartenstein and others, report similar cases of stomatitis aphthosa, diarrhœa, etc. Compare, also, the occasional reports in the publications on the subject of this disease by the German Official Sanitation Office. A collection of the cases of transmission of the aphthæ disease to human beings effected by the use of milk has been made by Würzburg.

Of the more recent cases, the following are worth attention: Fröhner described, in 1890, a case in an adult of transmission of the disease by fresh butter. Symptoms of fever appeared after 12 hours, and in 24 hours the under lip and chin were swollen and covered with vesicles, which were also found on the tip of the tongue. In 3 days' time the ears became covered

with vesicles, and erysipelatously reddened. Ptyalism and swelling of the parotid and sublingual glands were also present. The lesions healed in 12 days. In 1889, Weissenberg described a case in Berlin of a three-year-old boy who suffered from the disease, with symptoms of headache and pains in the limbs, considerable itching of the skin, vomiting, diarrhœa and rigors, with an outbreak on the third day of numerous vesicles on the lips, cheeks, tongue, tip of the nose and fingers. The entire eruption healed in 10 days. Siegel (1891) observed among several hundreds of people in the neighbourhood of Berlin a mouth epidemic, in the course of which 36 people died. He connected it with foot and mouth disease of cattle (?). Further special forms of affection occurring in human beings have been described by Heyne (ulcers occurring on the finger-nails and toes), Ebstein, Pott, Röder, Stierlin, Glaister, Bligh, and numerous practical veterinary surgeons.

Course and Prognosis.—The symptoms of foot and mouth disease vary greatly in different years. They are sometimes slight; at other times severe. The course is generally benign and typical and almost always ends in 2 or 3 weeks (mortality from 0 to 1 per cent.). At other times the course of the disease is so malignant that there is a mortality of from 5 to 50 per cent. in full-grown animals and of from 50 to 80 per cent. in sucklings, which frequently die suddenly during the first days after birth. Weak and badly-fed animals are more liable to die from the disease than strong and well-nourished ones.

The duration of an outbreak in a shed or herd is from 4 to 6 weeks. Usually it spreads very rapidly although its dissemination may be slow at other times. As a rule the animals regain their condition very quickly after recovery. In exceptional cases, however, convalescence may be delayed by the sequelæ of the disease, such as emaciation, decreased milk supply, even prolonged drying of the milk, retardation of conception, development of the symptoms of previously latent tuberculosis, chronic weakness of the heart, inflammation of the udder, chronic affection of the hoof, pruritus, cutaneous eruptions with loss of hair, blindness consequent upon deep-seated inflammation of the eyes, permanent lameness, gangrene of the extremity of the tail, etc. Besides loss of condition and decrease in the supply of milk, diminished fecundity also inflicts serious damage to owners.

Treatment.—Prophylaxis consists in segregating the infected animals and in using the milk only after it has been well [illegible]iled. A purely dietetic treatment generally suffices in cases [illegible]nign form of the exanthema; the animals being fed [illegible]dder or dry hay. They should have a constant supply

of pure drinking water and clean stalls. The floor should be kept free from moisture with dry litter, tan, gypsum or kainite. It is evident that medicines cannot cut short the course of the disease after it has commenced. Their action is essentially to be directed to symptoms, and is needed only in severe cases. More especially the intravenous injections of corrosive sublimate, recommended by Bacelli in 1901, have, as was anticipated, after numerous subsequent testings in Hesse, Prussia, Bavaria, Italy, etc., proved to be not only useless, but directly injurious (mercurialism). The ulcers on the mouth may be treated with disinfectants and astringent solutions (creolin, lysol, bacillol, pyoktanin, formaldehyde, alum, borax, tannic acid preparations, chromic acid, and nitrate of silver) ; the foot disease with watery solutions of creolin, lysol, pyoktanin, tar, carbolic acid, bluestone, etc. ; and the inflammation of the udder with ointments of creolin, lysol and camphor, and with salicylic or boric acid combined with glycerine. In symptoms of heart weakness, digitalis, camphor, alcohol, and atropin are given. The fever can be combated with antifebrin ; the diarrhœa with tannoform and opium. The treatment of the complications which affect the feet is surgical (tar bandages, shoes for the affected hoofs). As the experiences of the past decade show that foot and mouth disease, once having broken out, was not to be suppressed by the existing veterinary police regulations, the German Imperial Cattle-Plague Law of June 23, 1880, was altered in the direction of stricter observances in the new order of May 1, 1894. That foot and mouth disease cannot be suppressed even by those stricter police regulations has been demonstrated by the course of the plagues of 1896, 1897, and 1899.

Inoculation in Foot and Mouth Disease.—As early as the beginning of last century (Buniva), and frequently since then, inoculations have been successfully applied for the purpose of shortening the progress of the epizoötic in large herds, and for inducing the disease to run a mild and uniform course (Ercolani, Brauell, Renner, Hoffman, Wirtz, Spinola, Hertwig, Eewes, Brandes, and others). Not only is the induced disease, as a rule, mild and confined to the mouth ; but the practice of inoculation cuts short the duration of the epizoötic, and, as a consequence, the duration of the protective police regulations. We are, of course, considering the mild form of the disease ; for inoculation with the malignant variety would be too dangerous

to put in practice. The inoculation is carried out by placing some of the saliva of an infected animal into the mouth of the beast which is to be inoculated, after slightly excoriating the mucous membrane; by inoculating it anywhere under the skin with a lancet; or by drawing impregnated threads through the skin, preferably through that of the ear and tail. Pigs may be inoculated with a vaccinating needle on the snout. A rise of temperature takes place 24 hours after the inoculation, and aphthæ appear on the third day, burst on the sixth day, and heal about the tenth. Such inoculations made during the last few years in almost all parts of Germany have been generally attended with good results, especially when put into practice at the very beginning of an epizoötic. On an average the inoculations "take" in from 50 to 80 per cent. of the animals; the remainder proving to be immune.

Immunity and Protective Inoculation.—That foot and mouth disease confers a certain transitory immunity on the affected animals has for a long time been known to veterinary practitioners. The observations of veterinary practice have abundantly shown that several animals may remain immune for years against a new infection; while in others, on the contrary, the protection lasts but for a few months. Numerous cases have also been known in which no immunity occurred; on the contrary, many cattle having contracted this disease more than once in the course of a single year. In consideration of these facts, we have, in an earlier edition of our treatise, thrown doubts on the possibility of procuring immunisation by protective inoculation.

In the meantime new researches in this direction were inaugurated by the Commission for investigation of foot and mouth disease at the Institute for Infectious Diseases at Berlin, and at the Hygienic Institute at Greifswald (Löffler, Frosch, and Uhlenhuth) in the years 1897 to 1903. This Commission has established the fact, in harmony with previous veterinary experience, that in a number of the animals experimented on, an undoubted immunity was established after recovery from the disease; while others again sickened on repetition of the inoculation after an interval of some months, and then became immune. Accordingly, the Commission decided on a method of immunisation, by means of which, apparently, the vast majority of animals could safely be rendered immune to foot and mouth disease. This method consists of an intravenous

injection of a mixture of active lymph with the blood of immune cattle. A mixture of this kind made with blood serum of immunised animals (10–20 ccm.=160–320 minims), and virulent lymph from the vesicles ($\frac{1}{50}$ ccm.=$\frac{1}{3}$ minim), with the addition of 0.5 per cent. of carbolic acid, was recommended by Löffler in 1898, under the name of *seraphthin*, as a protective inoculation matter against foot and mouth disease.

The protective inoculation with seraphthin which was recommended by Löffler has, however, not proved satisfactory in practice. Seraphthin has not protected the inoculated animals from foot and mouth disease, but on the contrary, has distributed the plague among many of the inoculated herds observations of Leonhardt, Lothes, Collmann, Kitt, Hermann, Schmidt, Flatten, Schrader, Jonen, Friedrich, Schindelka, and others). On this ground the employment of seraphthin has been officially prohibited.

In 1900, Löffler and Uhlenhuth announced that they had succeeded in obtaining a serum from horses which protected sheep and pigs against foot and mouth disease for a period of from 3 to 8 weeks. A protection of so short duration, and limited exclusively to sheep and pigs, is obviously useless for practical purposes for the extermination of foot and mouth disease.

Finally, Löffler announced, in 1902 and 1903, that he had succeeded in obtaining a serum which protected cattle for an interval of 3 weeks against the natural infection, and thus was specially adapted to the prophylactic protective inoculation against infection incurred in cattle-markets, or from imported cattle. Nocard has also prepared a similar serum, but has pointed out that the short duration of the immunity (about 14 days), and the large quantities of serum required on the occurrence of an outbreak of the plague in a locality, are obstacles to the general adoption of the inoculation. These methods are, accordingly, of value only for agricultural shows and markets.

The problem of a reliable general protective serum-inoculation against foot and mouth disease of cattle remains, therefore, unsolved at the present day.

Protective Inoculation according to Hecker.—Experiments in immunisation were carried out by Hecker almost simultaneously with the researches of the Commission for the investigation of foot and mouth disease in the Institute for Infectious Diseases and in the Official Sanitation Department. The inoculation matter recommended by him was tested in the summer of 1899 on 1,000 head of cattle in the district of

Breslau, Frankfort and Potsdam, according to a plan arranged by direction of the Prussian Agricultural Ministry, after that formulated for the province of Saxony by the Chamber of Agriculture. In the official report published on October 20, 1899, the researches carried out on behalf of the State showed that Hecker's inoculation matter was not, from the nature of its composition, suitable for adoption as a curative or protective agent against foot and mouth disease. The mishaps which occurred (16 head of cattle became gravely ill after the inoculation; 12 had to be slaughtered, whilst 1 died) have also proved that it is necessary to submit every dose of the inoculation matter to a fresh testing before being used, in order to ascertain whether it has become unfitted for employment by the presence of toxic matter.

Whether the administration of potassium iodide (Pick) or of the *milk* of animals which have passed through an attack of the disease (Faber, Winckler) exerts an immunising effect is questionable.

VARIOLA.

A.—VARIOLA IN GENERAL.

Variola of man—Sheep-pox—Cow-pox—Horse-pox—Swine-pox—Variola of goats—Variola of dogs—Variola of birds.

History.—The variola of our domestic animals, like that of man, has been known for a very long time. Sheep-pox and small-pox of man are probably the only independent kinds of variola and probably have originally come from the East. We may point out that epidemics and epizoötics spread from east to west. Animal variola appeared for the first time in 1275 in England. Rabelais in 1578 was the first to describe it in France, in which country inoculation had been introduced at a very early period. Ramazzini reports its occurrence in Italy during 1691; and Stegmann, in Germany during 1698. Bourgelat recognised its contagiousness in 1763. The disease was spread to an extraordinary extent by protective inoculation, which was universally introduced at the end of the eighteenth century (1770 in Germany); by the importation of foreign, especially Spanish, sheep; and by improvements in the breeding of sheep. Thus in 1819 more than 1,000,000 sheep died of variola in France; and in 1823 nearly half a million in Austria. By the help of the regulations for the suppression of epizoötics, Germany is almost entirely free from sheep-pox, which still rages in Russia, Hungary, France, England,* and other countries.

* There has been no sheep-pox in Great Britain since 1862.—Tr.

Cow-pox has also been known in England for many centuries. Jenner in 1796 was the first to transmit it to man. He also proved that it conferred on men immunity to small-pox. Since that time, vaccination has become gradually established everywhere.

Horse-pox seems to have been of more frequent occurrence in former times than at present. Sacco and Jenner stated at the commencement of last century that they had frequently seen it. Jenner considered horse-pox to be the starting point of cow-pox. Röll states that horse-pox during 1855 in Vienna often occurred among young remounts. According to Bouley it is found more frequently than cow-pox in the neighbourhood of Paris. No case of horse-pox has been observed in Germany for several decades. In 1838, Hertwig inoculated himself and others with horse-pox, and thus produced on the hands and arms an eruption which closely resembled cow-pox. In former times, many cases occurred of blacksmiths becoming infected from shoeing horses which had horse-pox localised on the skin of the back of the pasterns. From this infection these men became immune to small-pox

Variola of Men has been much longer known than that of animals. It was recognised in ancient times, and spread, especially in the Middle Ages, over Western Europe, where, even during the eighteenth century, there was an annual mortality from it of nearly half a million. The practice of the inoculation of small-pox is exceedingly old, and has long been current in India and China. From thence it came through Asia to Constantinople, where Lady Wortley Montagu had her son inoculated in 1717. From Constantinople the inoculation of small-pox was first introduced into England, where Jenner proposed vaccination in 1798. From England it was brought to the Continent.

Occurrence.—Variola occurs in most of the domestic animals, such as sheep, cattle, horses, dogs, goats, pigs, as well as in man. It has also been observed in monkeys, camels, and buffaloes. It appears that variola is not an independent, primary disease in all species of animals. Old observers, such as Turner, Leroi, Ceely, Sunderland and Thiele, pointed out the genetic connection between cow-pox and small-pox. Roloff and Bollinger held the opinion that cow-pox derived its origin from small-pox. In fact, Bollinger maintained that small-pox and sheep-pox were the only two essential forms of variola; and that all the other kinds of variola (those of horses, cows, pigs, goats, and dogs) were adventitious forms of variola, which were respectively

carried from man or from sheep to these other animals. The similarity and close relationship of these various kinds of variola is proved by their reciprocal power of transmitting the affection, and of conferring immunity. Their respective identity with small-pox is amply demonstrated by the frequent interchanges made between small-pox and animal variola.

Chauveau, having failed to set up variola in cows from small-pox, disputed the connection between cow-pox and small-pox; and expressed his opinion that, although both kinds of variola may have had a common origin, cow-pox is not mitigated small-pox. On the other hand, Fischer, Pourquier, Ducamp, and others have succeeded in transmitting small-pox to cattle.

Etiology.—Variola is a contagious infective fever which manifests itself as an acute, typical exanthema. The contagium is both fixed and volatile. It is contained in the contents and crusts of the pustules, blood, secretions, excretions, expiratory air, and exhalations from the skin. In cattle it is usually absorbed by the organs of breathing or from the skin, when pustules are present. In the case of sheep-pox the virus can be carried for a distance of 220 yards. Chauveau has proved that the infective matter is contained in the solid and not in the fluid constituents of the lymph. Although the exact nature of the contagium has not been demonstrated, there is hardly any doubt that it is a bacterium. Zürn, Hallier, Cohn, Weigert, Klein, Toussaint, Semmer-Raupach, Plaut, Guttmann, Pfeiffer, Ruete, Guarnieri and other observers have attributed the disease to the action of micro-organisms. Guttmann and Grigorjew cultivated from the lymph a micrococcus, the staphylococcus cereus albus variolæ (micrococcus vaccinæ). Ruete believes that certain very motile diplococci are the cause. Pfeiffer, repeating the investigations made by previous observers, has called attention to the existence in the plasma of peculiar spherical formations, which have a homogeneous capsule and a finely granulated nucleus. They are from 20 to 30 μ long, sometimes enclose several protoplasmæ, and are stated to break up into spores (sporozoa). Nothing further is known respecting their connection with this disease. Van der Loeff, Guarnieri, von Wasiliewsky, Funk, and others found in the animal lymph, motile corpuscles, which they took for protozoa, and regarded as the proper causative vaccine agent, *sporidium vaccinale*; these so-called Guarnieri's corpuscles, nevertheless,

appear to represent merely specific degenerative conditions of the cells (compare chapter on Fowl-pox, Vol. II., p. 425).

The contagium of variola remains virulent for a considerable time; in fact it may continue so for some months when it is protected against decomposition. The contagium of sheep-pox remains active even in sheds for a month to 6 weeks, and is destroyed by putrefaction, high temperature, carbolic acid, etc. An attack confers immunity from subsequent ones. This immunity may be regarded as permanent, considering the short life of sheep.

General Symptoms.—The course of an attack of variola is usually divided into 5 stages, which in the typical course of the exanthema are prominently distinguishable, namely: incubation, initial period, eruption, suppuration, and exsiccation.

1. The *period of incubation*, that is to say, the time which intervenes between the entrance of the infecting matter into the body and the appearance of the symptoms of the disease, is about a week on an average.

2. The *initial stage* (*stadium prodromorum*), which lasts a day or two, is marked by fever, catarrhal affection of the mucous membranes, and erythema of the skin.

3. The *stage of eruption*, which continues from 6 to 8 days, manifests itself by the sudden appearance of red spots like flea-bites or pimples, which turn into firm red nodules the size of a pin's head and are surrounded with a red ring. They are chiefly found in the neighbourhood of the orifices of skin follicles. After a few days the tops of the nodules become pale and turn into bluish-white vesicles, which, especially in cases of inoculated variola, are depressed in the centre and are filled with limpid serous fluid. At this period the pocks are at their highest state of maturity, and are then most suitable for inoculation purposes.

4. During the stage of *suppuration*, which lasts 2 or 3 days, the contents of the vesicles become turbid and purulent; the vesicles change into pustules; and the depression in their centre disappears. The temperature, which had decreased during the stage of eruption, again rises.

5. During *exsiccation* the pustules gradually dry up into at first yellowish, and later on, dark-brown crusts or scales, which become detached and leave behind white shining cicatrices or brownish-red spots. The process of exsiccation goes on for 3 to 5 days.

Differing from the foregoing normal course of variola, several of the pocks may run together, and form large pustules (variola confluens), or the eruption may be complicated with a hæmorrhagic diathesis (variola hæmorrhagica), with gangrene of the skin (variola diphtheritica) in many cases.

General Remarks on the Anatomy of Variola.—If we follow the development of variola under the microscope, we shall see at first that the cells of the mucous layer above the papillary body swell and form here and there pale flakes which contain no nuclei. Afterwards, the papillary body transudes a serous fluid, and all the epithelial cells of the stratum mucosum dissolve with the exception of a few, which become drawn out into trabeculæ, and threads by the accumulated exudate. The vesicles thus form, between the papillary body and the horny layer, a cavity which is filled with fluid, and has trabeculæ and threads intersecting it. At the same time the papillary body and the cutis lying under it become inflamed, swollen, and infiltrated with round cells. The pustule develops from the vesicle by an abundant immigration of leucocytes from the papillary body into the fluid of the vesicles, and by the purulent breaking down of the septa. Healing takes place by the exsiccation of the pustule, and by the absorption of the cellular infiltration under the crust. The epidermis becomes replaced from the remaining tissue and from the edges of the sore.

The origin of the depression in the centre of the vesicle is explained in different ways. Some say that it is caused by the hair follicles and the orifices of the sweat glands, which lie in the centre of the pock* ; others, that the trabeculæ in the interior of the vesicle prevents the centre of the pock from rising up. It is also ascribed to the inflammatory infiltration of the skin at the periphery of the pock.

B.—THE VARIOLA OF THE SEVERAL DOMESTIC ANIMALS.

(1.) *Sheep-Pox.*

Etiology.—Owing to its well-marked epizoötic character, sheep-pox is of greater economic importance than the variola

* The idea here is that the hair follicle or sweat gland being firmly connected to the underlying true skin, prevents the epidermis at its external attachment from rising under the influence of the pressure exerted by the fluid contained in the vesicle ; and that, in confluent variola, the said connection with the true skin becomes destroyed.—TR.

of any other domestic animal. The infective matter is fixed as well as volatile, and possesses such vitality that it will continue virulent in sheds for as long as 5 or 6 months. Animals which have had the disease, whether naturally or by inoculation, will remain capable of transmitting it for 6 weeks after recovery. It is not destroyed by cold, and does not lose its virulence after having been kept at a temperature of 10.4° F. for two months (Nocard). It can, however, be speedily and effectually destroyed by diluted hydrochloric acid, 1¼ per cent. solution of carbolic acid, 5 per cent. solution of chloride of zinc or quinine, 10 per cent. solution of permanganate of potash (Grünwald), and by all the stronger antiseptics. The contagium chiefly spreads by infected, recently recovered, or inoculated sheep; or by intermediate bearers, such as shepherds, dogs, clothes, wool, skins, manure, fodder, railway-trucks, etc. During the course of the disease the milk is very virulent. Almost all ordinary sheep are susceptible to the contagium with the exception of newly-born lambs, which remain immune for a short time, or may even continue permanently immune if the ewe had been inoculated shortly before lambing. On the other hand, it is possible that lambs may be suffering from variola at the time they are born or the fœtus may die in the uterus in consequence of the infection. Algerian sheep (Chauveau) and Brittany sheep (Nocard) are more or less immune. According to Waldteufel, on the other hand, sheep-pox is stationary in Algiers; and confers immunity on the animals only after the transmission of the disease.

The variola of sheep may be transmitted either naturally, or by inoculation to cattle, goats, pigs, horses, and men. Schmidt and others have described a case of ordinary infection in man. Also, variola of man and cow-pox can be transmitted to sheep. The direct introduction of human variola into the blood in sheep produces a general eruption of variola (Küchenmeister and Tappe). On the other hand, the blood of variolous sheep is virulent at no period of the disease (Nocard).

Statistics.—During 1886, in Germany, 4,792 sheep became infected with variola, the chief centre of the disease being East Prussia (Mazovia), where the districts of Lyck, Oletzko and Lötzen were more especially affected. It passed also into one of the higher districts of Würtemberg. Germany has since then remained free from this disease, except in 1889, when a sporadic attack, which was quickly extinguished, occurred in 45 animals. During the years 1889–1899, Germany remained free from the disease. In 1900, it attacked 220 sheep in the district of Lüneburg (Bleckede), of which 2 died; the origin of the enzoötic could not

be ascertained. In 1901 the disease was distributed, apparently by Russian smugglers, in the Imperial districts of Königsberg and Gumbinnen (1,005 sheep were affected, of which 38 succumbed). In Austria, 18,000 sheep became infected during 1888. From 1877 to 1887, 0.5 per cent. of all the sheep in Austria were affected, with a mortality of one-third. In Hungary, in 1894, 16,000 sheep were attacked with variola; in 1897, 9,500; in 1898, 5,400; in 1899, 5,129; in 1900, 3,470; with a loss of from 7 to 16 per cent. In 1887, 17,000 sheep suffered from it in France; Roumania appears to be greatly plagued; in the 4 years from 1887 to 1890, nearly 150,000 sheep suffered.

Symptoms.—After a period of incubation of, on an average, 4 to 7 days, with a minimum of 2 or 3 days, the animal becomes feverish, has rigors, is depressed and weak, hangs its head, and ceases to feed and ruminate. The temperature rises to from 106° to 108° F.; the pulse is frequent, and the respiration accelerated. The conjunctiva is greatly congested and there is a slight serous discharge from the eyes and nose. A day or two later, red spots and subsequently red pimples appear in rapid succession on those parts of the skin which are free from wool, or which are only slightly covered with it, especially the head, neighbourhood of the eyes, nostrils, mouth, inner surface of the fore-limbs and hind-quarters, chest, abdomen, and lower surface of the tail. Less frequently the exanthema appears on the woolly parts of the body, and in some rare cases on the mucous membrane of the mouth and pharynx. As a rule, the eruption is not simultaneous; consequently different stages of the process may be seen close together on the same animal.

On the fifth day after the eruption, the pimples become pale on their summits and surrounded with a red areola. When the pocks lie in great numbers close to one another, as on the head and about the eyes, the neighbouring skin becomes greatly swollen. At this time the temperature falls, and a few days afterwards the vesicles increase in size; being sometimes raised, at other times flat. They then contain a limpid, colourless, or orange-coloured fluid. On the sixth or seventh day after the eruption, the pocks are ripe.

From this period the contents of the pocks become more and more turbid, and the vesicles turn into pustules; this change being accompanied by severe general catarrhal fever. The temperature again rises; the mucous membrane of the eyes, nasal cavity, pharynx, larynx and bronchi are catarrhally inflamed; a muco-purulent secretion flows from the eyes and nostrils; the animal slavers, regurgitates, coughs,

and suffers from difficulty in swallowing and breathing; diarrhœa sometimes appears; the head is greatly swollen; and the exhalations from the skin have a very bad odour. The pustules wither and dry up, the epidermis contracts, and the crust, which was at first yellow, assumes a dark-brown colour, and finally falls off, leaving a small depression in the skin, or a cicatrix, on which little or no wool will grow.

Under these normal circumstances the course of the disease lasts for about 4 weeks.

Course and Prognosis.—The course of variola may present many departures from the *normal type* just described. In some cases we find a very mild or even *abortive form*, in which the pocks are few and discrete, and the fever is slight. In other cases the morbid process does not go beyond the development of pimples, which peel off after a few days. Lastly, the eruption is confined to vesicles, with only a slight effusion of lymph and without the formation of pustules.

In *variola confluens*, which is a severe variety of the disease, several of the pocks run together so as to produce large pustules that give rise to extensive suppuration, great inflammation, swelling, and even gangrene of the skin, from which a fœtid odour is often exhaled. There is at the same time high fever; pocks are seen on the mucous membrane of the mouth, pharynx, larynx, bronchi, and even on that of the cornea; the respective lymph glands are considerably swollen and sometimes suppurate; the inflammation of the mucous membrane may sometimes assume a croupy character; and croupous pneumonia becomes occasionally developed. In unfavourable cases, symptoms of septicæmia or pyæmia with metastases in the articulations, serous membranes of the brain, etc., may appear. Or the patients may die from pneumonic sequelæ or from asphyxia, caused by laryngeal croup. Sheep take a long time to recover from an attack of this kind of variola, which gives rise to great exhaustion and cachexia. They sometimes lose their entire fleece, and often become chronically lame or blind.

In *variola hæmorrhagica*, simultaneously with the appearance of the eruption, hæmorrhages break out everywhere on the skin, mucous membranes, and especially in the pocks themselves. This hæmorrhagic infiltration of the pocks leads to their gangrenous disintegration (*variola diphtheritica*). Among other symptoms, hæmaturia is also observed.

The duration of variola in a herd may be very short, or may

be prolonged for months. The course of the complaint varies greatly in intensity. Only 2 or 3 per cent. escape infection, the mortality from which amounts to from 10 to 20 per cent. under ordinary circumstances, and to 50 per cent. or more under unfavourable ones. The prognosis is particularly unfavourable in cases of variola confluens and variola hæmorrhagica, and when the animals are either very old and weak or very young (sucking lambs). The percentage of losses may then be as high as from 80 to 90. Very hot or damp cold weather, bad food and unsanitary housing exercise an unfavourable influence on the course of the disease. Freshly-imported sheep become more severely infected than home-bred sheep, acclimatised sheep, and those which have been previously attacked and are consequently immune.

Not counting the direct losses from death, we find that there is a fairly large indirect loss from reduced production of wool, decrease in bodily weight, frequent abortion, and chronic morbid sequelæ.

Differential Diagnosis.—Sheep-pox may be confused with pustular eczema and urticaria of the head, especially when they are produced by certain fungi, such as rust-fungi and mould-fungi. If sheep-pox has not been observed for some years in a district, we may conclude that such sporadic pustular exanthemata are not variola.

Therapeutics.—The treatment of sheep-pox, like that of all other acute exanthemata, is purely expectant and dietetic. In complicated cases it may be necessary to apply a symptomatic treatment which can be carried out only with difficulty, or not at all, when a whole herd has become infected. Inoculation and veterinary police measures are the most important factors in *prophylaxis*.

Inoculation against Sheep-pox (Ovination).—*Inoculation* against sheep-pox, which, during the first half of last century and up to the end of the sixties, was practised as a prophylactic measure, even when no outbreak was threatening, has been found to be in every way objectionable. It not only artificially maintains permanent centres of the disease, but also spreads it, as has been well proved, from these foci to neighbouring healthy districts. In several countries, such as Prussia and Austria, the dissemination of sheep-pox went hand in hand with inoculation.

Inoculation is, however, to be recommended when the disease has already broken out in a herd; for it not only shortens the stay of the epizoötic, but also produces, as a rule, a milder and more local affection than ordinary sheep-pox. The mortality is often *nil*, and is usually not more than 2 per cent. Only exceptionally, and under very unfavourable conditions, does it amount to 10 per cent. Inoculation may be practised in healthy sheep which are in the neighbourhood of a severely infected flock, and are in great danger of catching the disease.

Technique of Inoculation.—The inoculation material, or "ovine," must be taken only from those sheep in which the course of the disease is benign, and the pocks fully ripe. The lymph should be as bright and clear as water, without any trace of turbidity or purulence. Consequently it is usually taken from the inoculated sheep 10 or 12 days after the inoculation, or 6 or 8 days after the eruption has appeared. The inoculated sheep should be segregated, and the sheep which are to be inoculated should not come into direct contact with the sheep from which the inoculation material is to be obtained, lest natural infection may take place simultaneously with artificial (inoculated) infection. The site of the inoculation is either on the inner surface of the ear, about 1½ inches from the tip (which position is not without danger owing to the proximity of the eyes), or, better still, on the lower surface of the tail, at about 4 to 4½ inches from the anus. In the latter case the animal must be thrown in order for the operation to be performed, which may be done with either a narrow and pointed inoculation needle, having a spoon-like hollow, or with an inoculation lancet. If time be not pressing when inoculating a whole herd, it is well to perform an experimental inoculation with from 6 to 12 sheep. The symptoms in inoculated sheep are those of a mild and regular attack of variola, with local manifestations restricted to the site of the inoculation and very slight general disturbance. The pocks become ripe on the tenth day after the inoculation. In exceptional cases the pocks do not appear on the site of the inoculation itself, but on the parts adjoining it. The eruption caused by the inoculation is followed, only in very rare cases, by a general eruption (secondary pocks). The after-treatment of inoculated sheep consists in protection against unfavourable conditions of weather and attention to diet. After 10 or 12 days the flock should be examined, and those animals which have not "taken" should be inoculated a second time.

In recent times several experiments have been made with attenuated (mitigated) inoculation material. Peuch attempted this by diluting the lymph with water (1 to 50–150); Nocard and Mollereau, by diluting it with water containing oxygen; Semmer-Raupach, by heating it to 131° F.; and Toussaint and Plaut, by cultivating it in nutrient solutions. Pourquier has found that inoculation with a pustule which has become developed from a previous inoculation with the ordinary virus of sheep-pox on the eleventh or twelfth day, confers immunity without giving rise to the eruption. He prefers the inoculation to be made at the end of the tail. In France, protective inoculation with the mitigated

Pourquier's inoculation matter is generally recommended. For production of large quantities of the pure virus Nocard recommends intracerebral inoculation. Recently the serum therapy has been successfully tried.

(2.) *Cow-Pox.*

Etiology.—Jenner's original idea that cow-pox sprang from horse-pox is nowadays held by very few. Even in former times, Turner, Leroy, Ceeley, Sunderland, Thiele and others, pointed to the connection between small-pox and cow-pox. According to Bollinger, however, the source of cow-pox is not small-pox, as was formerly supposed, but human cow-pox (variola vaccina). This view is strengthened by the fact that enzoötics of cow-pox often occur at the time (spring) when children are vaccinated; that cows have frequently been infected by human beings, especially by milkmaids who were recently vaccinated; and that human vaccinia can be easily transmitted to cattle. Besides cow-pox was frequently transmitted from affected cows to healthy ones, especially by milking; and sometimes, also, by purchased cows.

The contagium of cow-pox differs from that of sheep-pox, by being fixed and not also volatile; consequently, direct contact of the contagium, as in milking, with the injured skin is always necessary for the infection. According to Chauveau, Warlomont, Hugues, Straus and other French investigators, intravenous, intraocular, and subcutaneous inoculations confer immunity without producing either a local or a general attack of variola. The transfusion of vaccine blood from calf to calf gives immunity without any visible affection (Reynaud). Cow-pox can be transmitted to sheep, goats, horses and men; and human variola and vaccinia to cattle. According to Sunderland, Dinter, and Woodville, the direct inoculation of human variola or vaccinia generally confers immunity, and is accompanied only on rare occasions by an eruption.

Statistics.—From 1825–1868, 241 cases of cow-pox, and from 1873–78, 100 cases were reported in Würtemberg. In 1874, 374 cases; in 1877–78, 1,037 cases; and in 1888–89, 878 cases occurred in Denmark. As a rule, cow-pox is a rather rare disease.

Symptoms.—Cow-pox is chiefly found in young cows which have been recently put to milk. The eruption is generally confined to the teats and to the adjacent parts of the udder. Fever is either absent or only slightly marked. The development of the pocks takes place in characteristic and well-marked

stages. There is sometimes a slight disturbance of the general health of the animal, which is best shown by decrease in the quantity of the milk. The milk is thinner and of lighter specific gravity than usual. The implicated teats are somewhat sensitive, slightly swollen, and show, particularly at their base, isolated pink pimples, which subsequently change into vesicles. These pimples number as a maximum from 20 to 30, and vary in size from a lentil to a pea. The colour of these small and large vesicles varies according to the condition of the skin. They are bluish-white and nacreous when the skin is white and very fine; red and have a metallic lustre when the skin is light-coloured and thin; leaden-coloured and possessed of a metallic sheen when the skin is dark and thin; and cream-coloured and lustreless when the skin is thick and free from pigment. The vesicles have a depression in their centre; they are elliptical in form on the teats, and circular on the udder; and are surrounded by a red ring if the skin is not pigmented, and by a tumid, swollen and firm margin. They become matured about the tenth day, and are then the size of a bean. From that time they quickly become purulent, pointed, and dry up into a dark-brown, shining crust, which falls off in about 4 days and leaves a cicatrix. The pocks are frequently torn off during the process of milking. The duration of the eruption is about 21 days. Prognosis is so favourable that treatment is superfluous. Only in rare cases the eruption spreads over the whole body, and may then be found also on the head, inner surface of the thighs, buttocks, back, throat, chest, etc. In bulls it has sometimes been seen on the scrotum.

The course of the disease in the same animal is characterised by successive attacks, so that the respective pocks show different degrees of development. The affection spreads slowly from animal to animal in a shed, until all the cows, with very few exceptions, become attacked. Bulls, oxen, and young cattle are less frequently affected than cows. They sometimes receive the infection from the litter. Cow-pox usually lasts for several weeks in a shed.

Differential Diagnosis.—The eruption of cow-pox may be mistaken for that of foot-and-mouth disease, rinderpest, eczema of the pasterns from feeding on distillers' wash, mercurial poisoning, which may be set up by the use of mercurial ointments or inflammation of the udder, and false cow-pox or varicella (chicken-pox), the vesicles of which vary in size from a pea up

to a cherry, and, on drying up, leave behind them thin, paper-like crusts. In this respect, confusion may also exist with so-called variola verrucosa, which consists of a multiple non-contagious eruption of papilloma on the teats and which gradually disappears after having been present for months. Distinction of the true and false pox is sometimes difficult; as the typical development of the same is sometimes disturbed by the act of milking (Aronsohn); and because, as Ehrhardt has found, both false pox and true pox can be transmitted to man.

General Notes on Animal Vaccination in Man.—Since the time of Jenner (1798) the "original" lymph, namely, the true lymph of cow-pox or "vaccine" has been used for the vaccination of man. As, however, cow-pox is comparatively rare, and as its vaccine can therefore be obtained only with difficulty, "humanised" vaccine, that is, vaccination from man to man, was afterwards employed. On account of the possibility of the simultaneous transmission of human diseases, such as syphilis, animal vaccination has been recently re-introduced. Vaccination institutes for the purpose of animal vaccination were at first established in Milan, then in Paris (1864), Brussels (1868), and later on in all countries and large towns. For its cultivation, calves from 5 to 12 weeks old are generally used. The technique of the vaccination is very simple. The calves are fixed in the left lateral position on a table with the right hind leg drawn perpendicularly upwards. The skin from the symphysis pubis up to the umbilicus, and laterally to the fold of skin at the stifle, is shaved and carefully disinfected. Then, either "incision" inoculation or "surface" inoculation is performed: namely, we make either cross incisions about ⅜ of an inch long, or we inoculate the entire surface by means of from 50 to 200 punctures, and place on the incisions or on the punctures mature lymph from a previously inoculated calf. The vesicles become mature in 4 or 5 days, and the lymph contained in them is used for the vaccination of human beings and for further inoculation of calves. The inoculation material is obtained by means of a clamp forceps, with which the lymph can be easily squeezed out of the vesicle. One calf yields from 1,000 to 3,000 doses. The vaccine may be preserved in various ways. Capillary tubes are not to be recommended, because the vaccine, when kept in them, frequently becomes inert in as short a time as a week. A better plan is to keep the vaccine in a dry condition, which we may do by scraping off the lymph and crusts, drying them, and placing them between two glass plates, which are closed up with paraffin. In this way the lymph keeps well for months, and even years; or the lymph may be rubbed down in glycerine with the addition of an antiseptic, and then kept in capillary tubes. As an antiseptic, Röpke recommends: thymol, 1¼ grs.; spiritus, 8½ mins.; glycerine, 3½ oz., or acid salicyl, 4 grs.; aqu. destill. glycerine āā, 1¾ oz.

Animal vaccine gives quite as good results, as regards the effect and development of the exanthema, as human vaccine, to which it is greatly to be preferred. Animal vaccine can be produced in any desired quantity, and its use is free from the danger of transmitting human diseases. The

chance of transmitting tuberculosis by its employment need hardly be considered; for tuberculosis is very rarely met with in young calves, and no proof has yet been given that it can be transmitted by vaccination. Besides, we can obviate any such risk by a previous veterinary examination, and by the employment of tuberculin. We need hardly say that only healthy and perfectly unsuspected animals should be selected for the inoculation.

(3.) *Horse-Pox.*

Etiology.—Horse-pox, also known by various names such as: mallenders, "greasy-heels," etc., was formerly much more common than at present, and is now exceedingly rare. It occurs only in certain districts, for instance, in the environs of Paris, according to Bouley. It is probable that small-pox is the true origin of horse-pox, which is consequently identical with cow-pox and which can be transmitted to cattle and mankind. Inoculation of horse-pox in cows produces pocks similar to those of cow-pox and capable of producing the same protective effect in men. According to Hertwig and Pingaud, artificial as well as natural transmission of horse-pox to men (shoeing-smiths and grooms) produces in them an exanthema similar to that set up by cow-pox. Bollinger considers that the predilection shown by the exanthema of horse-pox to attack the skin behind the pastern, is due to the fact that that part is peculiarly liable to the fissures and other lesions of cracked heels and grease. In our opinion this liability may be more justly ascribed to the fact that that site is frequently touched by recently vaccinated shoeing-smiths and grooms, who probably often act as intermediate bearers of the infection. The udder of the cow forms the analogy to this.

Horse-pox rarely becomes general, a fact which Chauveau observed when he made his inoculation experiments with vaccine, intravenously and subcutaneously, by inhalation and by feeding. Warlomont and Pfeiffer obtained similar results.

The variolæ of sheep, cows, and man can be respectively transmitted to horses. Berger observed on two occasions natural infection of horses by sheep-pox. Pécus saw a transmission of cow-pox to the horse by means of cobwebs which were used to stop bleeding.

Symptoms.—The first symptoms are increased temperature, debility, and loss of appetite. We then notice on the flexor surface of the pasterns an erythematous exanthema which consists of congestion and swelling of the skin, and may,

spread upwards to a greater or less extent. At the same time the animal goes lame, and, if made to move, stumbles and lifts his feet high off the ground. On the inflamed part we find vesicles and pustules which soon burst and leave the skin highly congested, painful to the touch, considerably swollen, and covered with a viscid secretion. The surface of the affected part gradually dries up, or becomes covered with a scab, and the lesions heal in a few weeks. In rare cases the exanthema attacks other parts, especially those portions of the skin of the head in the neighbourhood of the lips and nostrils which are only thinly covered with hair; and the mucous membrane of the nose, mouth, and conjunctiva.

Differential Diagnosis.—Although the occurrence of horse-pox as a distinct disease is well established, its eruption has been frequently confused with that of other diseases, especially with the exanthema of stomatitis pustulosa contagiosa, which closely resembles it, except as regards the site of the attack. In France the two diseases are regarded as being identical. The fact that the eruption of stomatitis pustulosa can be readily transmitted by inoculation, even to cattle, makes a mistake all the easier. In our opinion, the two diseases have, etiologically, nothing in common. Grease and cracked heels may be confused with horse-pox, but can be easily differentiated from it by the fact that they do not manifest the typical course of variola, and that they cannot be transmitted by inoculation. The vesicular eruption on the genitals may possibly be mistaken for horse-pox.

(4.) *Swine-Pox.*

Etiology.—Swine-pox seems to be obtained chiefly from human beings (by means of bed straw), or from sheep, as for instance, on account of the animal occupying the same stall as an infected sheep. Young pigs are more often affected than older ones. Swine-pox can be transmitted experimentally to goats (Gerlach), and back from them to pigs. It can also pass in the ordinary way to **man.** In Hungary 517 pigs became affected with variola during 1891.

Symptoms.—Usually a general eruption breaks out over the entire body. There is fever, debility, loss of appetite, and congestion of the mucous membranes. Red spots (like flea-bites) appear on the head, throat, back, chest, inner surface of the fore

arms and thighs, abdomen, etc. These spots change successively into pimples, vesicles and pustules, which finally dry up, and leave black, concave, circular crusts. In exceptional cases we may notice an eruption on the oral mucous membrane, conjunctiva, cornea, and mucous membrane of the stomach and intestines. Often the disease varies greatly in intensity. In a few cases the eruption is accompanied by lobular, catarrhal broncho-pneumonia, ending in death (Csokor). Swine-pox has been frequently confused with urticaria, pustular eczema, and other diseases. Quasinsky inoculated young sucklings with sheep-pox and horse-pox lymph, without result; and on that account doubts the occurrence of variola in pigs.

(5.) *Variola of Goats.*

The rarity of caprine variola accounts for the fact that we find mention of only a few cases in veterinary literature. On the other hand, it appears to occur more frequently in Italy; in Norway, 264 cases occurred during 1890. According to Brémond, Bonvicini and Marcone, the variola of goats is a disease peculiar to these animals; because it cannot be transmitted to sheep by inoculation; and the cows kept in company with the affected goats never contract this form of the disease. Sheep-pox, however, cannot be transmitted to goats, either by inoculation or cohabitation. On the other hand, goat-pox has been experimentally transmitted to cattle by inoculation, without, however, producing general symptoms of the disease (Marcone). Fayet states that the goats which pasture with the sheep in Algiers are never affected with the pox; while sheep-pox produces heavy losses among the sheep. Nocard found that goats are immune to sheep-pox (negative intracerebral inoculation). The occasional cases of goat-pox appear sometimes in the general form of sheep-pox, as already described; at other times in the form of cow-pox, as an eruption on the udder. Hertwig states that goat-pox is characterised by its course being marked by successive attacks. In a goat-pox enzoötic observed by Zeeb, the general condition was not substantially disturbed in any of the animals (no fever, no loss of appetite); only the young kids, which had been infected in the mouth by sucking, displayed difficulty in taking their food.

(6.) *Variola of Dogs.*

Dog-pox can be transmitted experimentally from man to dog (Dupuis and others). In rare cases this may also occur,

accidentally (Weiskopf). We must view with suspicion many of the cases mentioned in veterinary literature; because this disease may have been confused with the pustular exanthema of distemper, or perhaps with mange and epizoötic aphthæ. In any case, dogs are very slightly predisposed to variola.

RINDERPEST (*Cattle Plague*).

History.—Although rinderpest was not known in classical times, it has probably existed for ages in the steppes of Eastern Europe and Central Asia. Its history is as ancient as that of the inhabitants of those regions. Rinderpest was brought for the first time to Western Europe by the migration of nations in the fourth century, and by the migration of the Huns from Central Asia, of the Alans from the Volga, of the East Goths from Southern Russia, and of the West Goths from Northern Hungary. Since then it has remained as a permanent resident in Western Europe. After its first appearance it was continually disseminated afresh over the countries of Europe by the many wars which took place, especially in the ninth century during the reign of Charlemagne, when Germany, in particular, suffered great losses in cattle. At the commencement of the thirteenth century, rinderpest devastated Eastern and Central Europe during the invasion of the Mongols. Rinderpest caused great havoc through Europe during the Northern War, the War of Succession, and the Seven Years' War in the eighteenth century. The first great epizoötic took place in the years 1709 to 1717, and spread from Tartary over the regions of the Don and Volga to Moscow, Poland, Hungary, Prussia, Austria, Southern Germany, Switzerland, Italy, France, Holland, and England. It is stated that one and a half million of cattle perished during the years 1711 to 1714. Ramazzini was the first to give an exact description of the disease, which he compared to variola. During his time veterinary police regulations were first instituted against rinderpest. Another epizoötic, which was described by Buniva, occurred during the years 1726 to 1734.

From the middle of the eighteenth century to the commencement of the nineteenth century, rinderpest was a permanent resident in all the countries of Europe, with the exception of Spain and Sweden, into neither of which any cattle were

imported. During the years 1740–1750, the loss of cattle was estimated at 3,000,000 head. From 1745–1752, it amounted, in Denmark alone, to 2,000,000. The total loss in Germany up to the end of the eighteenth century was reckoned at 30,000,000 head; and in Europe, at 200,000,000 head. The ravages of rinderpest were the chief reason for the founding of veterinary colleges, which began to spring up at that period. Many of the descriptions made at that time by Bourgelat, Bœrhave, Wolstein, Layard and others, are still extant. Lutz states that up to the year 1783 about 1,000 publications on rinderpest had been brought out. At that period protective inoculation against rinderpest was practised for the first time.

Inoculation was introduced into England by Dodson in 1744; into France by Courtivron in 1745; and after that into all other countries. In this connection, prominence must be given to the inoculations of Camper, Grashuis, and others in Holland, Older in Denmark, Bülow and Oertzen in Mecklenburg, Kersting in Hanover, Adami in Styria, Nebel in Hesse, Reich in Franconia, Sick in Prussia, Namsler in Silesia, Lorinser in Galicia, Pessina in Italy, Barrasch in Hungary, Welz in Würtemberg, Viborg in Denmark, Jessen, Raupach, Sergejew, Kobischow in Russia.

Rinderpest destroyed between three and four millions of cattle during the year 1792 in Italy, into which country it had been introduced by oxen which were intended to be used as food for the Austrian army. From 1795 to 1801 it raged in Southern Germany; and was widely spread by the wars of Napoleon (1805–1809), and by the wars of liberation (1813–1816). It invaded Europe from Moldavia and Wallachia during 1827–1828; and from Russia, Poland, and the Baltic provinces during 1830–1831 (Polish revolution). In 1844 rinderpest was introduced into Egypt from Roumania and Anatolia with a loss of half a million cattle. Russia lost one million cattle in 1844–1845. Prussia, and particularly Austria, both of which countries are on the frontiers of Russia, were visited by rinderpest nearly every year. For instance, Röll states that Austria lost nearly half a million cattle during the years 1847–1864. In Russia, the losses sustained in the years 1870–1880 amounted to £3,000,000; 1880–1885, £2,200,000; 1885–1890, £1,400,000; 1890–1894, £200,000. The wars of 1866 and 1870–1871 caused a wide dissemination of the disease. During the latter war 70,000 died in France, 30,000 in Alsace-Lorraine, and 10,000 in Germany. The last serious invasion of Germany occurred,

in 1878-1879, during which period 2,500 cattle died in Prussia, making a gross loss of £100,000; and 350,000 in Russia. We may say, speaking generally, that rinderpest has disappeared at least from Germany, thanks to the prohibition of import and the strict execution of the epizoötic regulations.

The last great epizoötic of rinderpest in Africa (1896 and 1897) involved South Africa (Cape Colony, Transvaal, Orange Free State, Natal), in which from 80 to 90 per cent. of the whole of the cattle of the country succumbed to the disease. In the Transvaal alone, 800,000 head of cattle fell victims to the plague and 250,000 were also shot; in British Bechuanaland, the losses comprised 1,250,000 cattle. In Turkey, three great plagues of rinderpest occurred in the years from 1896 to 1899; in Roumelia, from 70 to 80 per cent. of all the affected cattle died.

Etiology and Pathogenesis.—The infective matter of rinderpest is as yet unknown. All attempts made, both with microscope and cultures, to find specific micro-organisms have failed. The researches of R. Koch in South Africa have especially proved to be negative in results. Semmer assumes, probably with justice, that emigration of the infective matter, which has not yet been positively identified, into the body takes place through the respiratory apparatus, from whence it gains entrance into the blood, and sets up a general infection in it, only after secondary changes have become developed in the digestive apparatus.

The infective matter is both fixed and volatile. It is contained in the secretions and excretions, namely in fæces, urine, saliva, tears, and milk; mucous membranes of the nose, mouth, and eyes; sweat; expired air; blood; and various organs of the body. On the other hand, it is not to be found either in the gall (R. Koch) or in the blood-serum of animals suffering from the disease (Theiler). The infection may take place either directly through affected animals, or indirectly by intermediate bearers, such as manure, litter, straw, hay, soil, skins, wool, flesh, clothes, railway-trucks, ships, butchers, cattle-dealers, smugglers, dogs, sheep, birds, etc. It is characteristic of the contagium of rinderpest that it can be carried only for a short distance by the air, and that this distance varies inversely with the dryness of the atmosphere, and is consequently shortest in summer. The different accounts are somewhat conflicting, but still they go to show that the average distance

from which the contagium can be spread is probably 25 paces. It has often happened that in summer the transmission of the infection has been prevented by a ditch which separated the infected animals from the healthy. It thus appears that the contagium is rapidly destroyed in the air, and especially in dry air. According to Nencki and others, cattle suffering from rinderpest, and those wholly free from it, may for months remain in the same open shed with only a boarded partition between them reaching to the height of their heads, without any infection taking place. On the other hand, it remains virulent for a comparatively long time in fluids, and in the solid tissues of the body. If the nasal mucous which is used for inoculation be hermetically sealed up, it will remain virulent for 6 weeks, and in a few cases even for 9 months. The infective matter remains active in sheds for 4 months and in hay for as long as 5 months. Flesh retains its virulence after it has been buried for 3 months, and manure which continued in a frozen condition throughout the winter, proved infective after it was dug up in the spring. The intensity of the infective matter is severest at the commencement of an epizoötic. The virus is destroyed by desiccation if heated to a temperature of over 140° F. Theiler states that liquid blood loses its virulence if heated for a couple of days to from 96° to 104° F., or by being cooled down to a temperature of 5° F., by putrefaction, and by various disinfectants such as chlorine, sulphurous acid, carbolic acid, etc. It is easily destroyed by disinfectants.

Bacteriology.—Sanderson, Bristowe, Murchison, Beale, Semmer, Naczynski, Hallier, Klebs, Roschnow, Woronzow, Medwedski, Saweljeff, Metschnikoff, Gamaleïa, Sacharow, Tokishige, Simpson, Edington, Nicolle, Nencki, Sieber and Wyznikiewicz, R. Koch, and many other investigators have occupied themselves in the bacteriological study of the infective material of rinderpest, which, according to Semmer, cannot be demonstrated by the present methods of staining and cultivation. Semmer is of opinion that the only bodies which can be regarded as the cause of the disease are very small corpuscles, in the condition of spores, which can be distinguished only with difficulty, and which cannot be cultivated. He says that they exist in numbers from 1 to 6 in the enlarged cell nuclei, and that they are frequently surrounded by a bright areola. Saweljeff cultivated as infective matter sporulating bacilli, which break up into micrococci and streptococci. The colonies on agar-agar were at first greyish-white, then lemon-yellow, and finally red. The bacilli could be stained with methylene violet. Inoculations with these cultures produced rinderpest. The bacilli themselves are very motile, and have rounded ends. When found in the blood they may be straight or curved, and can be seen only with difficulty when unstained. Saweljeff believes that former investigators have seen the same bacteria

when they were in a different form of development. Metschnikoff describes a short bacillus of rinderpest with rounded ends which sometimes assumes the form of cocci, and which grows out into leptothrix-like threads. These bacilli are found chiefly in the ulcers of the abomasum, but are also met with in the blood. The cultures do not liquefy gelatine. Gamaleïa produced with pure cultures rinderpest in calves and guinea-pigs, but not in rabbits. Sacharow describes as specifically pathogenic a stout bacillus $\frac{1}{2}$ to $1\frac{1}{2}$ μ long; and Tokishige, a very small and short bacillus, the pure cultivations of which are stated to have produced the symptoms of rinderpest in experimental cattle. According to Nencki, Sieber and Wyznikiewicz, the infective agent in rinderpest is a minute body of 1–3 μ in diameter, which displays amœboid movements, is round in outline, of pale glistening aspect, and not staining by reagents. Nicolle and Adil-Rey have reported the preparation of cultures of the rinderpest microbe which was invisible under the microscope, and rapidly lost its power. They assume that it is contained in the leucozoa.

Occurrence.—Rinderpest is an infective disease peculiar to ruminants. Besides cattle, it can be transmitted to sheep, goats, deer, buffaloes, yaks, antelopes, camels, gazelles, aurochs, etc. As a rule, one attack confers life-long immunity. According to recent researches the calves brought forth by immunised cows are also immune (Kohlstock). Opinions vary as to the original home of rinderpest, which according to Sergejew and Semmer is the black earth district of European Russia (Southern Russia, neighbourhood of the Black Sea, and the country of the Don Cossacks). This district has a layer of humus a foot deep and luxuriant vegetation. Other investigators state that it originated in Asia, Siberia, the Kirghiz steppes, Tartary, China, Persia, India, etc., and consequently outside of European Russia. We at least know for certain that rinderpest never becomes independently developed outside the Russian or Asiatic steppes, and that every epizoötic of it in Europe has proceeded from those districts. Thus it was proved that the outbreak of rinderpest which took place in England in 1865, was caused by Russian oxen which had been imported by sea; and the epizoötic in Switzerland in 1866, by Austrian oxen, the infection of which was traced to the introduction of the disease from Russia. For this reason, Germany and Austria, which are on the frontiers of Russia, are continually threatened with rinderpest. The recent destructive outbreak of the plague in South Africa (Cape Colony, Transvaal), in the years 1896 and 1897, was apparently also due to imported cattle. Formerly the disease was spread by bullocks intended for slaughter during times of war; but is now disseminated by the transportation of cattle, traffic in

cattle, fairs, etc. Only in rare cases is it carried by skins, flesh, wool, butter, fat, etc.

Symptoms.—The symptoms of rinderpest are those of a severe, acute, infective disease, in which the digestive tract is the chief point of attack. The period of incubation is usually stated to be from 6 to 9 days. Roloff and others have, however, observed a rise of temperature in from 36 to 48 hours after infection. Raupach and Ravitsch found in their inoculation experiments, pronounced pathological changes in, respectively, 11 hours and 33 hours after inoculation. Semmer found the micro-organism in the blood and nasal mucus as early as 7 hours after inoculation. Taking into consideration that the first symptoms of the disease, of which increased temperature is one of the chief, may easily be overlooked; we may estimate the period of incubation at from 3 to 7 days.

Rinderpest manifests itself by certain preliminary symptoms which, as in the case of contagious pleuro-pneumonia, enable us to recognise the disease a few days before its obvious attack. At first the temperature rises to from 105.8° to 107.6° F. The fever is of the continued type, and displays but slight remissions, until other symptoms become developed, when it falls, as is the case in all other infective diseases. The pulse is small and beats from 60 to 120 in the minute. There is great debility, decrease in the yield of milk, and loss of appetite. The coat loses its gloss and stands on end, and the muzzle is dry. The function of rumination becomes disturbed, and the animal may have slight shivering fits.

After these preliminary symptoms, well marked rigors come on; the superficial heat is unequally distributed; the breathing becomes accelerated; and the visible mucous membranes (conjunctival; those of the nose, mouth, rectum and vagina) assume a scarlet colour, or manifest scarlet spots on their surface. There is entire loss of appetite; arrest of the function of rumination; thirst; constipation, with the fæces dry and covered with mucus; and sometimes slight colic. Later on a discharge, which at first is serous and subsequently sero-mucous, issues from the eyes, nose, and vagina; and also saliva flows from the mouth. The fæces gradually become thinner and thinner until finally violent diarrhœa, accompanied by colic, sets in. The evacuations are then fœtid, viscid, and sometimes mixed with blood. Frequently they are passed with considerable tenesmus,

and even with prolapse of the highly congested mucous membrane of the rectum. The animal becomes rapidly emaciated, staggers when walking, is very sensitive to pressure on the loins, and lies down a great deal. In exceptional cases the patient is highly excited, and even mad (congestion of the brain; the so-called nervous form of rinderpest). Others exhibit violent dyspnœa, and symptoms of severe inflammation of the lungs (coughing, rhonchi, and dulness on percussion; the so-called pneumonic form).

In the further progress of the disease, characteristic changes are to be seen on the mucous membranes. Red patches which may be flat or in the form of wheals, and quickly become covered with a greyish-white loose crust, appear on the mucous membrane of the lips, tongue, cheeks, gums, nostrils and vagina. The uppermost epithelial layer consequently becomes opaque, and yellowish-grey spots develop on it. Less frequently the crusts are formed from the tumours by their pulpy, caseous disintegration. The crusts on being shed, leave dark-red, hollow places (the so-called erosion ulcers), which readily bleed. In slight cases of the disease there may be no crusts or erosions. We may sometimes notice a similar eruption in the form of very minute pimples and pustules (which form an eschar) on the abdomen, inner surface of the thighs, perineum and udder, in which case it may be confused with that of variola. Pregnant animals frequently abort.

If the disease takes an unfavourable course, the emaciation and loss of strength become more and more increased; the animal keeps continuously lying down, grinds its teeth, and shivers. Discoloured, purulent, and fœtid discharges flow from the mouth, nose, vagina, and anus. The vagina and anus remain open; the temperature falls below normal; and the animal may die in convulsions, or may pass away very quietly. In some cases bloody froth is discharged from the mouth and nose before death.

Course and Prognosis.—In unfavourable cases, which are by far the most common, death usually takes place between the fourth and seventh day after the appearance of the first symptoms; and in particularly severe attacks as early as the fourth or fifth day. In the few favourable cases, recovery takes place slowly with a gradual decline in the intensity of the symptoms, so that the period of convalescence is somewhat prolonged. The progress of the disease in a herd or shed is rather slow and

proceeds in successive attacks, but increases in speed as it goes on. At the commencement, only a few neighbouring animals are attacked and then greater numbers, so that several weeks may pass before the entire herd or all the animals in a shed become infected.

The course of the disease varies greatly according to the character of the epizoötic and the breed of cattle. At the first outbreak of an epizoötic the disease runs a much severer course than towards the end. The grey cattle * of the steppes † of Southern Russia, Hungary, Roumania, Moldavia and Servia take rinderpest in a much milder form than other breeds ; for they possess, to some extent, hereditary immunity. As they often manifest the symptoms of the disease in a manner which is by no means well marked ; it is not always easy to recognise it in them. They frequently recover from an attack in 8 days. The mortality in ordinary cattle varies from 90 to 95 per cent. ; but in the grey steppe-cattle it is not more than from 30 to 50 per cent.

[As a result of his researches during the years 1899 to 1903 Dr. Lingard, who is the Imperial Bacteriologist of India, states that "In India and the East, rinderpest has been existent probably for hundreds of years, and it is extremely difficult to ascertain in such a vast extent of territory, what immunising effect may have been acquired by some of the cattle of this country. It is probable, however, that in some districts the cattle have received a certain amount of protection against rinderpest through their progenitors, but up to the present this has been impossible to estimate." We learn from him that the cattle in the plains of India possess a relatively large amount of immunity, but that the Hill (Himalayan) cattle are much more susceptible. He also tells us that the Aden breed of cattle "is most susceptible to rinderpest, probably as much so as Hill animals, on account of the isolated position of Aden, and the infrequency of the visitations of cattle disease to that portion of Arabia."—Tr.]

In *sheep*, as in *goats* and in the *camel*, the disease is milder, and its infectiousness is less than in cattle, although the symptoms are essentially the same. Among 4,000 affected sheep in Austria during the years 1859 to 1863, and among the same

* These Russian cattle vary in colour from dark iron grey to a light grey, cream or dirty white. They somewhat resemble the Mysore breed of cattle (the bullocks of which are largely used throughout India for draught purposes), except that they have no hump, and have thick curly hair on their foreheads. They are capital beef producers, but are poor milkers.—Tr.

† A steppe, in the Russian acceptation of the term, is an uncultivated plain, devoid of forest.—Tr.

number in Hungary from 1861 to 1863, there was a mortality of 66 per cent.; and one of 60 per cent. among 30,000 in Russia during 1878. Differing from cattle, sheep often suffer from pneumonic affections when attacked by rinderpest.

Rinderpest of Pigs.—Pluning states that he saw pigs in Sumatra suffering from rinderpest. The symptoms and course of the disease are said to have been the same as in cattle. On the other hand, Theiler could not produce rinderpest in pigs by inoculation with rinderpest blood.

Differential Diagnosis.—The correct diagnosis of rinderpest is often a matter of considerable difficulty at the first appearance of an outbreak, especially in cases which occur sporadically and in an unexpected manner; because rinderpest exhibits no symptom that cannot be found in other diseases. Consequently our diagnostic deduction must be made from the combined information obtained from symptoms, autopsy, progress of the epizoötic, and history of the particular case under consideration. The chief diagnostic symptoms are: high temperature; red spots and yellowish-grey coating of the mucous membranes, with subsequent formation of erosion ulcers, especially on the vaginal mucous membrane; mucous discharge from the mouth, nose, eyes, and vagina; grave intestinal symptoms; and excessive emaciation. The proof or the possibility of an infection is of the greatest importance. We should be careful not to confuse rinderpest with diseases which resemble it, especially with the following:—

1. The *malignant catarrhal fever of cattle*, which, however, is only very slightly contagious. It is generally stationary, runs a slower course, and is localised chiefly on the head and respiratory apparatus. Here the severe affection of the eyes must be carefully considered.

2. *Foot and mouth disease* shows a certain resemblance to rinderpest in the ulcerous changes in the oral cavity, in gastric attacks, and in the exanthema on the udder. The eruption on the buccal membrane is, however, very characteristic, and the exanthema is also found on the skin of the digits. The process is usually benign, and the disease spreads rapidly.

3. In *dysentery*, the diarrhœa appears earlier and is rather bloody. The intestine only is implicated, and the affections of other mucous membranes (mouth, eyes, nasal cavities, and vagina) are absent. The *post-mortem* appearances are also different.

4. *Mycotic inflammation of the intestines*, caused by various fungi, spoilt distillers' wash, etc., may possess great similarity to rinderpest in consequence of its severe course, and, at times, enzoötic appearance. Here, also, intestinal troubles and certain nervous affections are more prominent than in rinderpest. Besides, it is not contagious.

5. *Poisoning by virulent poisons and scalding*, caused by the partaking of hot distillers' wash, give rise to symptoms of corrosive gastro-enteritis, stomatitis, and pharyngitis. The resulting affections are, however, not contagious.

6. *Anthrax*, the gastric form of which may be confused with rinderpest. It runs a much more stormy course, and is not directly infectious. Its presence can be positively proved by *post-mortem* examination and by the microscopic demonstration of the bacilli.

7. *Pleuro-pneumonia*, with which rinderpest is sometimes complicated, can in most cases be distinguished with certainty by physical examination of the thoracic cavity.

8. *Rabies*, which might have to be considered in the nervous form of rinderpest, and *cow-pox* are easily recognised.

9. *Infectious hæmaturia* (hæmoglobinuria) of cattle may, according to Babes, be sometimes confused with rinderpest. It is, however, not contagious. R. Koch has observed in South Africa, complications of rinderpest and Texas fever. Janson states that in Japan the so-called "cattle influenza" may be mistaken for rinderpest.

Anatomical Conditions.—The pathological-anatomical changes in rinderpest essentially implicate the mucous membrane of the abomasum, small intestine, oral cavity, rectum, and vagina. The general changes which are also common to other infectious diseases are of less importance.

The cadaver is greatly emaciated, the thighs are soiled with fæces, and the neighbourhood of the mouth, nostrils, eyes, anus, and vagina is covered with a yellowish or discoloured muco-purulent discharge. Nodules and pustules are sometimes found on the skin, and especially on the udder. The mucous membrane of the mouth and the pharynx is congested in spots, swollen, covered with lumps of mucus, and exhibits rounded yellowish-grey caseous plates or deposits, which may be seen with the microscope to consist of detritus, cells, nuclei, and micrococci, and are caused by a superficial diphtheritic inflammation. The removal of the plates discloses ulcerous and highly-con-

gested depressions in the mucous membrane ; the so-called erosion ulcers.

These changes are best marked on the inner surface of the lips, lower surface of the tongue, buccal mucous membrane, and gums of the inferior maxilla.

In the *first three stomachs* the mucous membrane is congested in spots, and the epithelium is so loose that it can be easily detached. The contents of the rumen and reticulum are soft, and those of the omasum are often dried up, which is, however, a condition that is also met with in many other diseases. In some cases the contents of the omasum are fluid. Generally the abomasum is empty, and contains only a small quantity of a tough, muco-purulent, yellow, or sanious material. Its mucous membrane is highly congested, especially in the neighbourhood of the pyloric orifice. The congestion is partly diffuse, partly in spots, in the form of points or in streaks ; and its colour may be purple, cerise, or reddish-brown, with a tinge of slate-grey. The epithelium exfoliates. On the mucous membrane we may find small, brown-yellow caseous deposits, in the form of plates, which become detached in shreds, and leave behind indented, highly-reddened places, which are studded with blood points (erosions). The glands (peptic and mucous glands) of the stomach are swollen, and show considerable cellular hypertrophy.

Exactly the same changes are present in the *small intestine*, in which we find a good deal of swelling and congestion of the mucous membrane, with insulated, scab-like caseous deposits and erosions. In very severe cases these deposits form tube-like casts of the intestinal canal. At the same time there is considerable infiltration of the solitary glands and of Peyer's patches, which are swollen and prominent, and either undergo a purulent disintegration, in which case their contents will be discharged in the form of a plug if pressure be exerted on them, or they become covered with caseous or frequently purulent deposits, which are surrounded by a hyperæmic ring, and which, on becoming detached, leave ulcers behind. Peyer's patches often assume a sieve-like appearance, that is to say, they become areolated. Lieberkühn's glands are considerably swollen, and the intestinal villi cellularly infiltrated. According to Klebs, the entire mucous membrane is infiltrated with micrococci, which accumulate chiefly in the neighbourhood of the vessels, and sometimes occlude the lumen of the vessels.

the *large intestine* the inflammatory changes are much less l, and are greatest in the cæcum. Generally the

mucous membrane is merely swollen, congested in spots, is of the colour of slate or of the skin of an eel, and is covered with mucus. Sometimes the changes in the small intestine may be only of this nature, although in severe cases the entire mucous membrane of the small intestine down to the muscularis may become necrotic.

Frequently the *mesenteric glands* are greatly swollen and infiltrated, so that they resemble in consistency the medullary substance of the brain. In other cases these changes are absent. The *liver* has the appearance of clay, is discoloured, and soft. The *gall-bladder* is filled with thin gall in consequence of stenosis of the orifice of the biliary duct. The mucous membrane of the gall-bladder is swollen, congested, and may even be covered with grey-yellow [buff] plates of exudate. The *kidneys* show parenchymatous changes, are yellow-brown in colour, and abnormally soft. The mucous membrane of the urinary bladder is congested, and covered with mucus. Similar, and usually severer changes are seen in the mucous membrane of the uterus, vagina, and rectum.

The *nasal mucous membrane* is of a dark-red colour, and covered with grey-yellow soft scabs, after the removal of which the true tissue of the mucous membrane lies bare. Similar changes are found in the larynx and trachea, where the deposited masses are frequently purulent and of a creamy consistence. The lungs are sometimes hyperæmic and reddened in spots; sometimes œdematous and even hepatised, or emphysematous. Even pneumothorax and subcutaneous emphysema may be present. The *heart* is flabby, relaxed, and infiltrated with ecchymoses. The endocardium assumes at an early period a dirty purple colour. Hæmorrhages are found under the endocardium and epicardium; and a yellowish transudate in the pericardium. The *blood* is dark in colour, and coagulates with difficulty. There is a large increase in the number of the leucocytes, and the red blood corpuscles show various changes in form (poikilocytosis). Frequently the *nervous central organs* and their membranes are highly hyperæmic. An accumulation of red transudate is sometimes found in the cerebral ventricles and under the arachnoid.

The anatomical changes just described may greatly vary according to the character and the degree of the disease, and age, breed, and state of nutrition of the infected animal. In ordinary cattle the changes are always better marked than in cattle of the steppes, and in sheep. In South Africa, also, many

deviations from the European type of rinderpest were observed, (R. Koch, Theiler); especially the exanthematous and diphtheria-like changes in the mucous membrane of the mouth and gums were but slightly pronounced; while, in the intestine, at an early stage, extensive fibrino-sanguineous exudations appeared.

Therapeutics and Inoculation of Rinderpest.—The treatment of rinderpest is quite as unsuccessful as that of many other infective diseases which have a typical course. Besides, it is forbidden by the regulations for epizoötics on account of the danger of infection. Hence, affected animals must be immediately slaughtered. For certain districts, as the steppes of Russia and South Africa, inoculation may, however, be regarded as an important prophylactic agent, though only after an outbreak has occurred. Protective inoculation should never be practised, as it is very liable to spread the disease. The laws of all the European States, with the exception of Russia, forbid inoculation, even after the disease has broken out; because pole-axing is much more effective, and because the mortality (about 36 per cent.) caused by the inoculation of ordinary cattle is extremely high. In the cattle of the steppes it amounts only to about 10 per cent. Favourable results of inoculation are most frequent towards the end of the epizoötic.

The method of inoculation is varied. We have to differentiate between the older subcutaneous method of inoculation, and the serum-and-bile inoculation lately introduced by R. Koch.

1. *Older method.* The technique of the older, subcutaneous method is simple. A clean sponge is introduced into one of the nostrils of an affected animal, and is allowed to remain until it is saturated with nasal mucus. It is then removed, and its contents are squeezed out into a small glass, which is closed up. A drop of this inoculation material is then injected subcutaneously on the neck by means of a Sticker's syringe. Even in Russia, where there were four inoculation institutes, inoculation has fallen more and more into disrepute.

2. *Koch's method* (serum-inoculation, bile-inoculation). In his researches in quest of the infective agent of rinderpest in South Africa (Kimberley), Koch found that both the blood-serum and the bile of cattle which had passed through an attack of the plague were possessed of immunising properties. The serum inoculation requires 100 c.cm. ($3\frac{1}{2}$ oz.) of blood-serum; its influence is of brief duration, and confers but a passive immunity. It serves, however, to isolate a plague-free locality from an

infected one by a broad girdle of immunised territory. The bile-inoculation is more effective, and is carried out simply by subcutaneous inoculation on the healthy animal with 170 mins. (10 c.c.) of the bile of cattle suffering from rinderpest, or which have passed through an attack. From this bile-inoculation, after 10 days, an immunity results which lasts 4 months. The local effect consists solely of a hard swelling of the size of a fist, which disappears after some weeks. The bile-inoculation is, above all, of very great value in infected localities. Kohlstock, who introduced the Koch method of inoculation into German South-West Africa, recommended a double inoculation, with bile and with rinderpest blood (secondary inoculation).

Kolle and Turner recommend the simultaneous injection of 17 mins. (1 c.c.) of virulent rinderpest blood on one side of the body, and the subcutaneous injection of 4¼–11¼ drs. (15–40 c.c.) of serum on the other side (simultaneous method). Koch's bile method has the disadvantage that the animals remain susceptible to the infection for a whole week after the inoculation. Further, 3–7 slaughtered cattle, which either are sick of rinderpest or have passed through the disease, are used for the purpose of inoculating 100 head of cattle with their bile. Also, the mixture of bile with glycerine, which was adopted by Edington, is unsuitable, as the addition of the glycerine lowers both the degree and the duration of the immunity. According to Hutcheon, the bile-inoculation method of Koch has been completely abandoned in Cape Colony, on account of its great imperfections, and supplanted by the serum-inoculation method of Kolle.

Nencki, Sieber and Wyznikiewicz (Russia) have endeavoured to perfect the method of immunisation with blood-serum, by a gradual raising of the proportion of the pest virus. They have also confirmed the observation of Kolle, that large doses of anti-pest serum may even cure rinderpest when used in the first two days of the fever; and found that Koch's bile-inoculation varied greatly in its effects on account of the quality of the bile. Bile of green colour, taken from cattle slaughtered from the fifth to the seventh day of the fever, generally produced, without further reaction, a passive immunity lasting from 3 to 5 months. Sometimes however, this bile produced a fatal attack of rinderpest. Yellowish-red or blood-red bile (of animals which had died) nearly always produced a fatal inoculation illness. Accordingly, the bile-immunisation should be adopted only when the anti-pest serum fails. Tokishige (Japan) has also carried out experiments with the view of increasing the immunising power of rinderpest serum.

Theiler (Transvaal) found that the serum of rinderpest animals did not contain the infective matter; and, also, that the anatomical conditions of rinderpest in South Africa presented some variations. The serum of cattle (which had passed through the disease) produced also in animals already affected a beneficial influence; and the milk of immune animals appeared to possess a curative power. Blood-inoculations have had no result either in horses, dogs, pigs, cats and guinea-pigs; nor in dogs, pigs and poultry which had been fed on infected meat. The international

South African Congress held in Pretoria in 1897 has expressed the opinion that Koch's bile-inoculation generally conferred but a temporary immunity; and that some animals, after the bile-inoculation, showed fatal symptoms. The bile-inoculation can, therefore, in the greater number of cases, not give absolute protection; but it can be regarded as a means of checking the course of an enzoötic. Koch's method has consequently been greatly modified by the Boers in the Transvaal (more than 170 mins. (10 c.c.) of bile-inoculation repeated every 3 months; the mixing of various kinds of bile, subsequent injection of virulent blood). In spite of the many unfavourable results, the risk of the bile-inoculation, when suitably carried out, is, according to Theiler, for the farmer, very small; and the inoculation is, therefore, a means of saving him in great measure from losses. Theiler states that in herds which are already infected, favourable results are more rarely obtained, though such results are possible. The Congress has, therefore, recommended that serum, and not bile-inoculation, should be adopted in infected herds. On the other hand, the practice of slaughtering and of quarantine cannot be successfully adopted in South Africa. In the Transvaal 150,000 head of cattle were said to be successfully inoculated. Theiler states that serum-inoculation has been generally adopted in South Africa, and that rinderpest has, by its use, lost much of its terrors. The serum is now obtained in this way: the immune ("salted") cattle are first, for the purpose of increasing the immunity, again inoculated with 3½ oz. to 17 oz. 5½ drs. (100–500 c.c.) of virulent blood; and, from 4 to 8 weeks afterwards, blood is withdrawn from them for the purpose of obtaining serum. The seats of inoculation for the serum are specially the subcutaneous tissue behind the shoulder, and the front part of the breast. The more serum injected, and the earlier the injection is carried out on affected cattle, the greater is the success. In sheep and goats, and also pigs, Theiler could never produce rinderpest by inoculation of rinderpest blood.

Rogers (Calcutta), in accordance with the instructions of the Indian Government, tested the various forms of inoculation which had been employed in South Africa. Koch's bile-inoculation produced, in the cattle of the lowlands and plains, an immunity of about 4 months duration, whilst it failed with the cattle of the hills. Kolle-Turner's simultaneous method proved reliable, and had good results with the cattle of the lowlands and with buffaloes. For the very susceptible cattle of the hills, Rogers recommends the simultaneous method, with subsequent blood-inoculation (Rogers' modification).

[Dr. Lingard has had admirable results with the inoculation of anti-rinderpest serum during the year 1902–1903. In Bengal, the death rate from inoculation with serum was 1·09 per cent. (112 in 10,190); in the Punjab, 0·0019 per cent. (4 in 23,861); in the provinces of Agra and Oudh, 0·015 per cent. (2 in 1,353); and in Burma, 0 per cent. (0 in 4,006). As there was an outbreak of rinderpest in Bengal at the time of the inoculation, "the total deaths of 112 in 10,190 animals injected with serum, instead of being an argument against the utility of the protective serum, is strongly in favour of the good work accomplished by it, for in any outbreak of the disease, whatever moment may be selected for the injection of the animals, a certain number must always have the contagium of the disease incubating in their systems" (*Lingard*). Dr.

Lingard points out that the cattle of the Punjab and Burma have a high degree of natural or acquired immunity.

During 1897, Koch and Edington have, more or less successfully, carried out extensive protective inoculation experiments against rinderpest with infected bile. The most hopeful results, however, appear to be obtainable from the method of Danysz, Bordet, and Theiler, a translation of whose *Report on Rinderpest to the Transvaal Government* may be seen in the *Veterinary Record* of the 20th February, 1898, to which number of that periodical I beg to refer my readers for full details. These observers at first confer comparative immunity, by the injection of immune blood, on experimental cattle, to which they then transmit rinderpest by natural infection, with the result that these partially protected cattle take the disease in a mild form, and, on recovery, become immune for the remainder of their lives. In this, these pathologists act on the knowledge that the injection of blood serum obtained from cattle which have recovered from rinderpest, renders the experimental cattle capable, for a short time, of withstanding the effects of the disease more easily than they would otherwise do; although it does not prevent them from taking the infection. In this method, defibrinated blood is used in preference to blood serum; as it is more economical in practice, and is at least equally effective.—TR.]

TEXAS FEVER—PIROPLASMOSIS.

Etiology and Pathogenesis—*Texas Fever* (Texas fever, splenic fever, Texas plague, "red water") is a disease of cattle which occurs epizoötically in North and South America, especially in Texas, Kansas, Missouri, Louisiana, Jamaica, the Argentine ("tristeza") and Venezuela ("lomadera"); also in Africa (German East Africa, Cameroons, Cape Colony, Transvaal, Algiers) and in Australia; in 1894 it became known in Germany by the fact of its presence having been proved in American cattle that had been imported into Hamburg during that year. Smith and Kilborne have furnished us with a good deal of exact knowledge concerning its pathology. According to them, Texas fever is an infective hæmoglobinæmia which is caused by the presence of blood parasites in the red corpuscles. These hæmatozoa have received the name of "pyrosoma (piroplasma) bigeminum."* These blood-parasites are of animal nature (hæmatosporidia, sporozoa); and are found as round, egg-shaped, or pear-shaped corpuscles of from 2 to 4 μ in length

* The parasite of Texas fever is termed *Piroplasma bovis* by Drs. Stephens and Christophers (*The Practical Study of Malaria*). It is from 2 to 4 μ in length, and from 1 to 2 μ in width.—TR.

and from ½ to 2 μ in breadth, usually in pairs, in the interior of the red blood-corpuscles (in the primary stage of existence their form is rod-shaped, curved, ring-shaped, or of willow-leaf outline).

The infection of cattle takes place through the agency of cattle ticks (*boophilus bovis s. rhipicephalus annulatus*). These ticks infest the skin of the cattle in great numbers, and communicate the piroplasmata to them through numerous minute cutaneous lesions. The development of the ticks takes place, according to the facts ascertained by the agricultural research station in Baton-Rouge (Louisiana), in the following manner: The pregnant female ticks allow themselves to drop from the skin of the cattle to the ground, where they lay from 1,500 to 3,000 eggs, from which the six-legged young escape in from 15 to 20 days. The young ticks now adhere in masses to the highest points of the blades of grass and of the shrubs, and with outstretched fore-legs await the cattle, on the skin of which they take a firm hold with their mandibles. After being from 12 to 15 days on the skin, they are sexually mature, copulate, and drop from their host, after having, without change of position, filled themselves with blood. Koch has proved that young ticks which have never come into contact with sick cattle, but have descended from ticks which lived on sick cattle, may in their own turn produce Texas fever.

The cattle born in localities in which Texas fever prevails as an enzoötic are immune, despite the presence of numerous ticks. In foreign imported cattle a single attack of the disease confers immunity. The fact that ticks living on immune cattle are able to produce Texas fever in imported cattle is also noteworthy.

Symptoms and Anatomical Conditions.—The disease occurs in two forms, one acute (midsummer), the other chronic (autumn). The most important symptoms of the disease are high fever (105° to 107° F.), hæmoglobinuria, alteration in the form of the red blood-corpuscles (macrocytes, hæmatoblasts, poikilocytes, hydræmia, anæmia (decrease of the number of blood-cells from 5–8 millions per cubic millimetre blood, to from 1–3 millions), loss of appetite, stoppage of rumination, decrease in the secretion of milk, constipation alternating with diarrhœa, dyspnœa, and rapid emaciation. The disease sometimes runs a fatal termination from exhaustion in 4 or 5 days; in other cases, not till 14 days, or even later. Relapses

are not rare, and the disease may last for months. The mortality is especially high in summer; but the autumnal chronic form is milder. The anatomical changes are: the presence of pyrosomæ (5 to 50 per cent.) in the red blood-corpuscles; poikilocytosis; anæmia; a thin and watery condition of the blood; hæmoglobinuria; hæmorrhagic nephritis; and hyperæmic swelling of the spleen. Besides this, we find ecchymoses in the heart; gelatinous swelling of the connective tissue in the neighbourhood of the kidneys and portal vein; enlargement of the liver; anæmia and biliary engorgement in the brown-yellow liver. According to Koch there occurs a very characteristic filling of the bile capillaries with a yellow glistening solid mass, and diffuse reddening of the gastro-intestinal mucous membrane.

Diagnosis.—The microscopical demonstration of the blood parasites is best conducted with a magnifying power of from 800 to 1,000 diameters. In living cattle a drop of blood is procured from a puncture or a small cut in the skin. Make a preparation of the blood or of the organic juice in the usual way adopted for the staining of bacteria. The drop of blood must be placed as quickly as possible on the cover-glass, and spread out at once into a thin film by means of the edge of a second cover-glass. After allowing the preparation to dry in the air, we draw it through the flame, as in the examination of bacteria. Smith considers it better to expose the cover-glass for 1 to 2 hours in dry air at a temperature of 230° to 248° F. After this, the layer of blood is stained with a solution of Löffler's methylene blue, either by putting a few drops of the solution on it, or by floating the preparation for ½ to 2 minutes on this solution in a glass capsule. We can also simply use a watery solution of methylene blue; but this method will take more time. The preparation is then washed in water, dipped for one moment into a 1 per cent. solution of acetic acid, and washed again with distilled water. The cover-glass can then be placed with the adhering drop of water on the object-bearer for examination, or it may be mounted in Canada balsam. Lately, the Romanowsky staining method has been recommended. In examination of the fresh blood without staining, the drop of blood must be brought rapidly on to the object-glass, with a drop of physiological salt solution; the cover-glass is at once applied, and surrounded with a paraffin ring (bright, vacuole-like spots in the blood-cells).

[Lingard terms Texas fever *Hæmoglobinuria* or Red Water in cattle and buffaloes, and gives the following synonyms: *Tristeza* (Argentina), *Tick fever* (Australia), *Lal peshab* (Urdu), *Rokto mootra* (Bengali), and *Rodsyge* (Norway). For further remarks on diseases conveyed by ticks, see Vol. I., p. 565.]

Therapeutics.—The simplest prophylaxis of Texas fever in infected localities is to disinfect the pastures and to remove all cattle from them for at least one year, at the end of which period all the ticks will have died of hunger. This result is also obtained by the cultivation of the infected prairies, or grazing sheep, as the cattle-ticks cannot live on the latter, owing to the excess of oil in their wool. Housing has the same effect.

To prevent the introduction of Texas fever into localities previously free, by means of immune cattle infested with ticks, they have in America employed thorough dipping with a layer of petroleum, paraffin oil, mineral oil, creolin-like cresol compounds, fish oil, cotton-seed oil, and other fatty oils, which kill the ticks rapidly. The cattle are driven into large tanks, which are filled to over the height of their bodies with water, covered by a layer of oil of several inches in thickness. Specially recommended is also a bath of 875 gallons of petroleum (dynamo oil), to which 94 lbs. of sulphur has been added. In Africa (Rhodesia) they have recently used warm arsenical baths in dipping basins.

As animals which have passed through the disease are known by experience to remain immune to Texas fever, protective inoculation has, accordingly, been employed as a preventive measure against this disease. In America, blood-inoculation of young cattle has been recommended, and produces a milder form of Texas fever with subsequent immunity (85 to 170 mins. (5–10 c.c.) of the blood of cattle which have passed through the disease, and are accordingly immune). This blood inoculation, in connection with the successive placing out of the inoculated cattle on infected pastures, allows the disease to run a mild course, attended with comparatively little danger (maximum loss of 8 per cent.). On the other hand, the serum inoculation has not proved satisfactory in America.

Piroplasmosis in South Africa.—According to Theiler, there exists in South Africa, besides the piroplasmosis of cattle (Texas fever, "red water"), a corresponding one of the horse (equine malaria), and of the dog (canine malaria). 1. The piroplasmosis of cattle occurs as the usual "red water" and the more recent "Rhodesian red water" ("East Coast red water," "East Coast fever," "South African fever"). The

ordinary "red water" occurs in a benign, subacute, and a malignant, acute form (imported cattle; mortality of from 60 to 90 per cent.). In very acute cases, red urine appears at the onset of the disease; and the animals die suddenly at the end of two days, after the development of nervous symptoms (staggering, convulsions). In cases of longer duration, extreme emaciation appears, with vesicles on the mucous membranes and also jaundice. The subacute form manifests itself by gastric disturbances (dry, blood-stained fæces; diarrhœa). The East Coast "red water" begins, after a 10 to 12 days' incubation, with a sudden attack of high fever (up to 107.8° F.). In spite of this the cattle display a good appetite till 2 days before death. The symptoms consist of recession of the eyeballs; foaming at the mouth; dyspnœa; bloody, tar-like diarrhœa; rapid emaciation, and swelling of the lymphatic glands. Red urine and jaundice are usually absent; recovery is rare. 2. *Equine malaria*, as a rule, presents acute jaundice, with hæmorrhagic staining of the conjunctiva; great emaciation; polyuria; marked yellow staining of the urine; nervous symptoms and coma. 3. *Canine malaria* usually runs an acute and fatal course, with high fever, jaundice, lassitude, loss of appetite, vomiting, yellow fæces evacuations and rapid emaciation; the duration is of 2 or more days. In the chronic form, general emaciation appears; pronounced anæmia; vomiting and diarrhœa.

The *African Coast fever*, or *Rhodesian fever* (Rhodesian "red water"), is not, according to Koch, identical with Texas fever (ordinary red water), but forms a special disease; which is also caused by piroplasmata, with ticks (*rhipicephalus decoloratus*) as intermediate hosts. The piroplasmata of African Coast fever are, however, much smaller, and also of different form than those of Texas fever. In contrast to the latter, also, few of the red corpuscles are destroyed, for which reason neither anæmia nor hæmoglobinuria appear. Coast fever holds, apparently, the same relation to Texas fever that ordinary malaria does to tropical malaria (distinct malaria parasites, *i.e.*, piroplasmata).

Piroplasmosis of Dogs.—Piroplasmata have recently been shown to be also the causative agents of disease in dogs; first, in Italy (Piana, Galli-Valerio); then in East, West and South Africa (Koch, Mautoux, Robertson, Theiler and others); and, lastly, in France (Almy, Nocard, Motas), where the disease seems to be of frequent occurrence. As in cattle, so also in the dog, the disease is conveyed by ticks (*dermacentor reticulatus* in France; *hæmophysalis lœchi* in South Africa). Hunting dogs are usually affected (ticks). The piroplasmata of dogs morphologically resemble those of cattle, but are of a different species, and cannot, therefore, be transmitted to cattle, horses, goats, sheep, etc. On the other hand, they are easily transmissible to dogs by subcutaneous, intravenous, and intramuscular inoculation. For young dogs, a single drop of blood suffices for a fatal infection. After a single attack of the disease, permanent immunity remains. The piroplasmosis of the dog appears in two forms: the acute, which always leads to death; and the chronic, one which usually terminates with recovery. 1. In the acute form, the dogs show depression; loss of appetite; fever, with a temperature over 104° F. for from 2 to 3 days; then a sudden fall below the normal, to 91.4° F.; anæmia; jaundice (not constant); rapid, thready pulse; rapid respiration; staggering; and, finally, complete paralysis. The urine is, from

the beginning, albuminous; often reddish, deep-red, or even black (hæmoglobinuria); it may contain as much as 3.5 per cent. of hæmoglobin (the spectroscope shows the two absorption bands of oxyhæmoglobin). The blood is pale, as if mixed with water, and shows a diminution of red blood corpuscles from 7 to 2 millions per cubic millimètre; microscopically, deformed shapes of the red blood corpuscles are seen, especially macrocytes. 2. The chronic form is characterised by persistent anæmia and muscular weakness; fever, jaundice and hæmoglobinuria appear occasionally. In the acute form, the *piroplasma canis* is very easily found in the blood; in the chronic often for the first time only after several days' repeated examination. According to Kitt, the most suitable method of staining is the following, which was devised by Nocard and Almy: The spread-out preparation, which has been treated with alcohol-ether for at least one hour, and of which the fluid constituents have been allowed to evaporate, is placed in a bath of the following composition. Three solutions are prepared: 1, a 0.05 per cent. of Höchster's eosin (marked extra BA); 2, Nicoll's carbol-thionine; 3, Borrel's blue (Borrel blue with silver oxide), a saturated solution. These solutions are filtered separately. We then take 3 f. dr. of the eosin solution, and add to it 17 mins. of the carbol-thionine, and 2 drops of Borrel blue. This mixed solution must not again be filtered. The spread-out preparation is placed on the surface of the coloured solution, so as to float thereon (a minute raft); and is allowed to rest in that position for at least 4 hours (even up to from 12 to 24 hours). It must then be carefully washed in running water, and treated with Grubler's orange-tannin, which is let fall in drops on the preparation, for a space of half to one minute, and then washed again; finally, it is dried and embedded in balsam. The orange-tannin enables us to differentiate the blood-cells from the parasites, and clears the field of precipitates. The parasite appears, in blue colour, with nucleus of a vivid carmine-red tint; the blood-cells are of an orange-red.

Carceag of Sheep is an epizoötic hæmoglobinuria allied to Texas fever. It occurs chiefly in the delta of the Danube, and is caused, according to Babes and Starcovici, by a blood parasite (hæmatococcus). It is also supposed to give rise to hæmoglobinuria in cattle in Roumania. Betegh also found the parasites in the eggs of ticks. Ziemann states that a similar disease occurs among sheep also on the island of St. Thomas. The disease of sheep known in England as "louping-ill" is also apparently identical with carceag (McFadyean). The symptoms consist of rigors, fever, colic, hard bloody fæces, anæmia, and, in rare cases, of hæmaturia. By *post-mortem* examination we find the hæmatococci in the red blood corpuscles, hæmoglobinuria, swelling of the spleen, intestinal hæmorrhage, an anæmic condition of the muscles, and a yellow gelatinous œdema in the subcutis. A similar disease, produced by hæmatozoa, has been described by Perroncito in Sardinia under the name *Proteosi*; by Bojoly in Algiers, under the name of Ferulose; and also by Bonome, Williams, de Mia, and others.

MALARIA—INTERMITTENT FEVER.

Malaria in man is a pure miasmatic infective disease which occurs everywhere except in the Polar regions, and particularly in swampy regions (Roman Campagna, Pontine Marshes, Sicily, Hungary, the countries of the Lower Danube, and the tropics). Laveran attributes its occurrence to the presence of protozoa (malaria amœbæ). Its chief symptom is an attack of fever* which generally lasts for only a short time, and possesses a strikingly regular type (daily, every second, every third, or every fourth day, etc.; febris quotidiana, tertiana, quartana, quintana, etc.). The spleen, as a rule, becomes considerably swollen. The fever varies greatly in type, and may take the form of pernicious, intermittent fever, remittent and continuous malarial fever, chronic malarial cachexia, or masked intermittent fever with neuralgia. The chief remedy for malaria is quinine.

More recent investigations by R. Koch have shown that the malaria parasites are transmitted to man by stinging gnats (mosquitoes), culex pipiens, anopheles maculipennis, etc.). They form an independent group of protozoa, not identical with the piroplasma bigeminum of Texas fever. They are hæmatozoa, to which, in addition to the parasites of the quartan, tertian, intermittent and tropical fevers in man, the malaria-like parasite of monkeys, and also two parasites in the blood of birds belong, *i.e.*, the proteosoma Grassi, and the halteridium Danilewskyi. Koch thinks that the hæmatozoa found in cattle and bats may also belong to the true malaria parasites. The development of the malaria parasite commences in the red blood corpuscles (young, half grown, full grown parasites). They then separate themselves from the blood corpuscles and differentiate into male and female, after which they impregnate in the stomach of an intermediary host, the stinging gnat, and then evolve further (transformation into tiny worms, coccidia-like balls, ripe crescent-shaped germs in the poison-gland). Through the sting of this intermediary the young parasites pass into their ultimate host (man, animals).

* Lingard states that the majority of horses in the Karnal Remount Depôt (India) suffer from fever of an intermittent type.—Tr.

[The following remarks on malarial fever of man have been taken from the Translator's *Veterinary Notes for Horse Owners*:

"The 'plasmodium of Laveran,' which occurs in different forms, gains entrance into the body by means of mosquitoes of the genus *anopheles*, that act as its carriers. This malaria parasite is a protozoon, and not a plasmodium, which term, in this application, is incorrect, though sanctioned by use.

"This parasite, in the form of very minute needle-shaped organisms, is injected into our blood along with the saliva of the mosquito, at the moment this fly bites our skin. Each of these spores (to adopt Professor Lancaster's nomenclature) enters a red corpuscle, undergoes a stage of development (amœbula), and breaks up into numerous spherical spores (enhæmospores) which enter the blood by the destruction of the red corpuscle. They then invade and destroy other red corpuscles and, by their continued multiplication, infect the whole of the blood. Other stages of development take place, until their final transformation in the blood is attained. They are then in a suitable state to be sucked up by mosquitoes, and after that takes place, they arrive in the stomach of their carrier, undergo changes and migrations, and at last gain access into the mosquito's salivary glands. They are then ready to make a new invasion. We thus see that these organisms are parasites of the mosquito, as well as of man; and that a human being who is suffering from malarial fever, can infect the mosquitoes of a place which had hitherto been free from that disease. In this way, one human being can indirectly infect another human being. Direct infection from one man to another man can be communicated only by inoculation. Mosquitoes have their regular meals, just after sunrise and after sunset, which are consequently the times for specially avoiding these insects.

"The constitutional disturbance is chiefly due to the destruction of the red corpuscles and to toxins elaborated by these parasites (Manson). 'Observations by Koch, confirmed by Stephens and Christophers, have recently shown that in intensely malarial districts, practically all the young children have malaria parasites in their blood. As the native children get older, their blood is progressively less liable to the infection, and in native adults the parasites are rarely found. Immunity from malaria can therefore be acquired' (*Manson*).

"There are several forms of malarial fever, of which intermittent fever (ague) and remittent or continued fever are the most common.

"Dr. Manson points out that the successive generations of the parasites of intermittent fever tend to become simultaneously mature in the blood of their human host, about the same time every day, every second day, or every third day, according to their species. Thus, those which live for 24 hours, produce quotidian (daily) fever; those which exist for 48 hours, tertian fever; and those whose cycle of development is 72 hours, quartan fever. I have not seen similar paroxysms and remissions in the malarial fever of horses.

"The bilious symptoms often seen in cases of malarial fever are chiefly due to the bile of the patient containing an abnormally large amount of *bilirubin*, which is the bile pigment that gives a yellow colour to the skin and other tissues in jaundice. As bilirubin is produced by the decomposition of hæmoglobin, it follows that the more free hæmoglobin there is in the blood, the greater will be the quantity of bilirubin in the bile. In malarial fever, the blood becomes loaded with free hæmoglobin, owing to the destruction of red corpuscles by the malarial parasites.

"The chief breeding places of mosquitoes are shallow pools which are not large enough to contain fish, and which do not dry up between showers. Hence the best way to rid a district of mosquitoes is to drain or fill up these pools, or to destroy the mosquito larvæ by pouring into these pools a sufficient quantity of paraffin oil to cover their surface; and to kill all the mosquitoes within reach. A large number of the mosquitoes which infest houses, can generally be found resting on the walls in the interior of these buildings during the day.

"Colonel A. H. Morris, who is in charge of the Northern Territories of the Gold Coast, reports: 'I caused all holes which might contain puddles, and so become breeding-grounds for the anopheles mosquito, to be filled up. Some hollows in rocks containing about 18 inches of water, were discovered filled with thousands of larvæ. The Hausas' and Carriers' lines were inspected twice a week in order to insure no stagnant water being allowed to remain in old pots or tins. The general result has been an immense reduction in the number of mosquitoes.'

"The great importance of shallow pools for the breeding of mosquito larvæ is due to the fact that such pools do not contain fish, which would devour the larvæ.

"Equine malarial fever is widely distributed throughout South Africa, where it is generally known as 'biliary fever,' on account of the lining membrane of the eyelids assuming a bright yellow colour, and the mucous membrane of the lips becoming tinged with yellow. The mortality is small."]

Malaria in Animals.—The question whether human malaria is transmissible to animals has been answered by Koch in the negative. Numerous experiments regarding its transmissibility which have been made on anthropoid apes (ourang-utangs, hylobates), have proved unsuccessful. He, therefore, on that account considers it incredible that animals further removed from man in the scale of creation should shelter human malaria parasites.

On the other hand, many cases have been described in veterinary literature of malarial diseases which were for the most part produced by piroplasmata. Kolle observed a disease of cattle in South Africa ("bovine malaria") with markedly remittent fever; protozoa were found in the blood, which displayed a striking similarity to the malaria parasites of man. The "equine malaria" described by Theiler in South Africa in 1901 belongs, according to his most recently published views (1903), to the domain of piroplasmosis (see Vol. II., p. 637). Malarial diseases in horses and cattle are repeatedly met with in the Roman Campagna, a principal focus of human malaria, and are often successfully treated with quinine. Celli and Dionisi, however, believe that they are not produced by the malaria parasites of man, but by hæmatozoa, which display a great

similarity to the piroplasma bigeminum of Texas fever. In the Sudan also (Pierre), in Senegambia (Dupuy), in Tunis (Berard), in Russia (Popow), and also in other countries, malaria-like diseases are met with in horses.

On the other hand, pure malaria occurs in birds (Grassi, Danilewsky, Koch). There have hitherto been found two blood parasites (halteridium and proteosoma). Halteridium is found in the characteristic dumb-bell shape in summer, also, in Germany, in the blood of nestlings (pigeons, sparrows, finches, tree-falcons). The proteosoma has hitherto been observed only in southern countries; in canaries, robins and crossbills. Both these malaria parasites produce a disease which either runs an acute course, accompanied by grave general symptoms, or a chronic course without fever.

THE TSETSE, OR SURRA, DISEASE.

Nature and Occurrence.—The epidemic disease known in Africa as Tsetse, or Tsetse-fly disease (Nagana), and in the East Indies as Surra, principally attacks cattle, horses, asses, mules and camels. It is a malignant anæmia caused by blood parasites (trypanosomes). Its existence in Africa was first proved by Bruce in Zululand; it occurs in East as well as West Africa, in the Congo and in the Niger districts, and in Algeria. In German East Africa it is, with Texas fever, the most deadly of all animal diseases. In many of the valleys adjoining the rivers no cattle can be kept, or even transported through them. R. Koch found the disease in Dar-es-Salam and on the island of Mafia; but according to his investigations, the real sources of the disease are at the foot of the Usambura and Pare Mountains, on Lake Victoria and the Ruaha River. In addition to the animals already mentioned, buffaloes, sheep, goats, antelopes, dromedaries, hyenas, elephants, dogs, cats, monkeys, rabbits, rats, mice and hedgehogs are also susceptible to the disease, while zebras, native sheep, goats and game are said to be immune. Human beings are not liable to it. In Europe the existence of the disease was first proved in 1903 in the Berlin Zoological Gardens in a pony imported from Togo.

Etiology and Pathogenesis.—The Surra disease, as in intermittent and Texas fever, is transmitted by the sting of flies, particularly by that of the Tsetse fly; the specific blood

parasite trypanosoma Brucei being carried from diseased to healthy animals through the intermediary of the Tsetse fly. The trypanosoma Brucei belongs to the group of spermatozoa, from 26 to 33 μ in length, and from 1.5 to 2.5 μ in breadth, colourless animalcules, in shape like a fish or worm, provided with a flagellum and an undulating membrane 2 to 3 times as long as the diameter of one of the red blood corpuscles, among which they actively move. They propagate by longitudinal division, and in chronic courses of the disease occasionally disappear from the blood of affected animals, and seem to develop further in the lymph glands, in the spleen and in the red marrow. They are not identical with the trypanosomes so frequently occurring in rats.

The Tsetse fly—*glossina morsitans*—is an insect widely distributed in South and Central Africa, and its devastating effects on the cattle of those regions were known to African travellers from the time of Livingstone. It is particularly found along the river courses in the hot and damp lowlands with forest, shrub, and cane vegetation. It is nearly ½ inch in length, and haunts shady places (under the leaves and between the hairs), and springs on animals and men. The sting of the blood-sucking insect is painless; in half a minute it has sucked its fill of blood. If it subsequently stings another animal it transmits the trypanosomes even after an interval of 48 hours. No other method of infection takes place under ordinary circumstances. Outside the animal body the trypanosomes live in blood only a short time, from 1 to 4 days. The congelation of the blood does not injure the parasites. The trypanosomes can be readily transmitted by inoculation (cutaneous, subcutaneous, intravenous, intraperitoneal) to mice, rats, guinea-pigs, rabbits and dogs. Dogs in subcutaneous inoculation show the parasites in the blood after from 2 to 3 days. Transmission through the genital membrane (coïtus), the eye-lid ligaments and the membranes of the stomach and intestines (feeding) has no results, nor is an intra-uterine infection of the fœtus possible.

Symptoms and Course.—After an incubation stage of from 9 to 12 days (Koch), the trypanosomes appear with symptoms of fever, in the blood (investigation of a suspended droplet, with the admixture of a solution of salt; staining with methylene-blue in spread-out preparations). The animals either succumb with increasing weakness, poverty of blood and emaciation; or they pass into a chronic state of ill-health.

Recovery appears never, or but quite exceptionally, to take place in cattle. On dissection, we find the appearances of pernicious anæmia ; œdematous, gelatinous swelling of the subcutaneous tissue of the neck, belly and extremities; turbidity of the cornea ; extreme emaciation ; swelling of the spleen, and sometimes also of the lymphatic glands ; also numerous ecchymoses.

Inoculations have shown that horses and asses die several weeks after infection ; dogs and cats in from 2 to 3 weeks ; and rats in 2 weeks.

Treatment.—The Tsetse disease has hitherto been regarded as incurable. Quinine and arsenic especially have been used without result. All experiments in the way of immunisation, with blood, serum, bile, etc., have proved failures. A spontaneous extermination of the Tsetse fly has lately been observed in South Africa. This was caused by the destruction of all the herds of cattle (with the eggs of the flies) as the result of rinderpest.

Surra.—The Indian Surra disease, which was first investigated by Evans in 1880, is produced by the trypanosoma Evansi, which is apparently identical with the trypanosoma Brucei of the Tsetse fly disease. The transmission is carried out by the blood-sucking tropical gad-fly (*tabanus tropicus* and *lineala*—in Hindu, burra dhang). The Surra disease occurs especially in horses, mules and camels. The general appearance of the disease resembles that of the Tsetse fly disease (anæmia, emaciation, œdema, fever).

Cadeiras Disease (*Disease of the pelvis*).—This is a disease observed by Elmassian among horses in the interior of South America. It is likewise produced by a trypanosome (Elmassiani), and displays great similarity to the Tsetse fly disease (transmitted by the Brazilian stinging fly, *stomoxys calcitrans* (?). Its specially distinctive symptoms are paresis of the hind-quarters and hæmaturia. Cattle appear to be non-susceptible to this disease. The trypanosoma Elmassiani is distinguished by the absence of the centrosoma of the other trypanosomes.

Trypanosoma Equiperdum.—The trypanosome which has been found in dourine is distinguished from the other trypanosomes by its minuteness, and its power of passing through mucous membranes. On this account, the view of the identity of dourine with the Tsetse fly disease, which was formerly advanced, cannot now be maintained. (See Vol. II., p. 505.)

BRADSOT (BRAASOT, BRAXY) OF SHEEP.

Etiology and Occurrence.—By the term bradsot,* braasot or braxy (" the quick disease "), is indicated an anthrax-like, peracute and extremely malignant hæmorrhagic inflammation of the abomasum in sheep (gastromycosis ovis), which has long been known on the west coast of Norway; in the Danish isles, Iceland and the Faröe Islands; in Sweden and Scotland; and also in Mecklenburg. First pointed out by Krabbe, and then fully described by Nielsen and Jensen, this enzoötic disease appears in the countries named only in the harvest and winter months; especially in young sheep, while those of three years old and upwards are seldom affected. Nearly all cases of sickness in sheep (from 5 to 50 per cent. of the total number) fall victims to braxy. In 1870, 11,000 sheep died of it in Iceland; the yearly loss in Scotland amounts to 150,000 sheep. According to Peters, this is a more serious plague in Mecklenburg than anthrax.

According to Nielsen, braxy is caused by the *bacillus gastromycosis ovis*, which measures from 2 to 6 μ in length, and about 1 μ in thickness; it is anærobic, and stands in close relationship with the bacillus of quarter-ill; in contrast to the latter, however, it is pathogenic to pigs, mice, pigeons and fowl. It is not, however, identical with it, as braxy inoculation confers no immunity against quarter-ill (Jensen). It is strange that sheep cannot be infected through the alimentary canal, by feeding on the bacilli of this disease. Subcutaneous infection produces œdema and muscular inflammation.

* Bradsot, which is an infectious disease, is well known in Norway and Scotland. In North Britain it is termed "braxy." It occurs only during autumn and winter; is peculiar to sheep; and is confined almost entirely to sheep not over 3 years of age. It is almost always fatal, and runs its course in a few hours. The chief symptoms are: great depression; unwillingness to move; desire to remain recumbent; swelling of the hind-quarters; tympanites; and sometimes frothing from the mouth. We find, *post-mortem*, a dark purple discoloration of the abomasum from hæmorrhagic infiltration; more or less congestion and degeneration of the abdominal organs; dark colour of the blood: and rapid decomposition of the dead body, accompanied by a considerable evolution of gas. The flesh can be used with safety as human food. For a summary of C. O. Jensen's article on this subject, see *Veterinarian*, September, 1896.

It is probable that ovine anthrax is not unfrequently mistaken for braxy in Scotland.—TR.

Symptoms and Post-Mortem Conditions.—The sheep usually fall down, as if struck with apoplexy, and die in the course of a few hours ; in less rapid cases, a striking lassitude is observed, and even deep coma. On dissection there is frequently found a hæmorrhagic inflammation of the abomasum, with œdematous and hæmorrhagic infiltration of the mucous membrane; also blueish hæmorrhagic patches appearing through the serous coat, in the walls of the abomasum. We sometimes find larger parts of the intestine similarly affected. In rare cases local changes in the stomach and intestine are absent ; and there is a general infection, with marked enlargement of the spleen and parenchymatous swelling of the internal organs.

Therapeutics.—According to Nielsen, protective inoculation (subcutaneous injection of dried kidney substance) on the inner surface of the thighs has proved prophylactic against braxy. Tokishige has established experimentally that goats, sheep and horses can easily be immunised against braxy; and that the blood serum of the immunised animals, as well as the milk of the small ones experimented on, confers passive immunity.

Reindeer Plague.—This is a disease, described by Lundgren, which prevails in an extremely destructive form among the Lapland reindeer of North Sweden ; and is produced by a specific bacterium, and is characterised by a peracute course, and by the presence of œdema of the head and abdomen. The infection takes place, apparently, through wounds of the skin and intestinal mucous membrane. A new experimental work on reindeer plague and reindeer plague bacilli has been published by Bergmann.

Scarlet Fever of man was formerly regarded as a disease which also affected animals. It is evident that this idea arose from petechial fever having been mistaken for scarlet fever. It is strange that this old fallacy should have recently again cropped up by the assertion that scarlet fever of man is produced by the milk of cows suffering from scarlet fever. Klein even states that he has found a micrococcus scarletinus both in the scarlet fever of man and in the milk and suppurating ulcers of cows suffering from scarlet fever, and that he has transmitted the disease to calves by inoculations of cultures of this micrococcus. He also asserts that he has found this micrococcus in condensed milk, and that he has inoculated it successfully in calves and mice. As might have been expected, these statements of Klein have not been confirmed. We may point out that cows are absolutely immune to human scarlet fever, the correctness of which statement has been proved by Crookshank, M'Call, McFadyean, Axe, Edington, and others. On the other hand, cows' milk, like many other things, may serve in a few cases as an inter-

mediate bearer of the contagium of human scarlet fever. The supposed cases of scarlet fever in the horse and in the cat do not appear to have been very well demonstrated. Marek has described a disease (scarlatinoid) of the horse closely resembling scarlet fever.

Cholera.—It is uncertain if animals under the natural course of events suffer from cholera. Haffkine asserts that Asiatic cholera has occurred in pigeons and geese simultaneously with the appearance of the disease in man. Ogata states that he has observed in a dog in Tokio a case of Asiatic cholera with all the characteristic symptoms, and that he demonstrated the comma bacillus in it. Pfeiffer and Gruber state that cholera can be produced experimentally in guinea-pigs by the intraperitoneal inoculation of young cultures, but without any intestinal affection. According to Haffkine, an intravenous inoculation of specially prepared cultures of cholera produces in rabbits a disease which is very similar to cholera, and which is accompanied by "rice-water" evacuations from the intestine. Koch succeeded in causing an infection in guinea-pigs by means of cholera comma bacilli, after having artificially produced a disturbance in the activity of the abdominal organs, and neutralised the hydrochloric acid of the stomach with a solution of soda. Gamaleïa states that he has transmitted the cholera bacilli to pigeons; according to Rindfleisch and Walther, however, this can be effected only by intramuscular injection of highly virulent cultures.

Febris recurrens, the relapsing fever of man, has up to the present been transmitted by inoculation only to monkeys (spirilla in the blood). Steel states that he has observed among horses in India a disease in which he found in the blood motile spirilla.* The disease was transmitted to monkeys and dogs by inoculation. Gabritschewski and Cantacuzene have observed a septicæmia (fever, diarrhœa) arising from spirilla (spirochætes) in the blood of geese.

Yellow-fever is stated to attack horses and dogs in America and Sicily. According to Saranelli, the bacillus of yellow fever (bacillus icteroides) is pathogenic to domestic animals, especially dogs (vomiting, hæmorrhages nephritis, gastro-enteritis); while birds are, on the other hand, immune.

[In *The Lancet*, 19th March, 1904, Sir Patrick Manson states: "As regards yellow-fever, although we do not know the germ of the disease, we know how the disease is acquired. The germ is so minute that it passes through the closest filter and seems to be ultra-microscopic, but we know that it exists, and that, like the germ of malaria, it is transferred from one victim to another by a certain kind of mosquito, the common West Indian tiger mosquito (*Stegomyia fasciata*)." Sir W. K. Rynsey (*The Lancet*, 9th August, 1902) tells us that "in Havana, yellow-fever was endemic for a century and a half. During the past year it has been freed from the scourge by killing the mosquitoes in the neighbourhood of each focus of disease as discovered, and by carefully disinfecting

every house that had lodged a yellow-fever patient, in order to destroy the mosquitoes that had bitten a sick person."—Tr.]

Measles.—Measles does not occur in our domestic animals. Arnch professes to have observed a case in the dog. Supposed cases in pigs were really those of erysipelas, or of some other disease. Behla states that he succeeded in transmitting measles to swine. Josias claims an equally successful result with 5 monkeys. Geissler has, on the other hand, failed to transmit measles to sheep, pigs (sucklings), goats, dogs, and cats (14 negative results).

Beri-Beri, which attacks mankind in the Dutch East Indies, is an infective neuritis multiplex peripherica. Eyckmann states that it is found in monkeys and fowls, with symptoms of paralysis, in consequence of degeneration of the peripheral nerves. Lacerda reports that it is also met with in pigs and horses. Lellmann has described a similar case occurring in the dog. Glogner attributes the disease to amœbæ which he says he has found in the red blood corpuscles.

Milk sickness.—Kimmel describes under this name a "milk disease" which occurs in the centre of the United States of America in cattle, sheep, goats and horses, and lasts from 2 to 8 days, with loss of appetite, constipation, excitement, rigors, etc. It is stated that human beings who drink the milk of such infected animals, suffer from a similar disease.

African horse-death.—(South African Horse-sickness). This is known in South Africa under the names of "Perreziekte," "Paardenziekte," or "horse-sickness." Sander, who investigated it in German South-West Africa, considers that this disease in its two forms (Dunpaarden ziekte and Dikkoppaarden ziekte) is identical with anthrax. Rickmann described horse-death in South-West Africa, and Edington in Cape Colony; both hold the view, in opposition to Sander, that horse-death is not anthrax. According to Rickmann, it is a disease similar, or closely related, to pernicious malaria of man; and which, however, has nothing in common with piroplasmosis. Theiler investigated both forms in the South African Republic, and described them as distinct diseases; a micro-organism cannot be demonstrated in cases of horse-death by any of the methods of research known up to the present time. More recently, Theiler has distinguished 4 varieties of horse-death: peracute (septicæmic) acute (pulmonary); subacute (cardiac); and chronic (atypical). The average duration of the disease is from 5 to 6 days; the mortality is 90 per cent. McFadyean and Nocard regard horse-death as a form of septicæmia, which is produced by a nocturnal flying insect.

Karassan.—This name has been given to a disease which, according to Wediornikoff, occurs among the steppe cattle in Russia, and which is probably identical with anthrax.

Akpaipak is a disease which is seen in the steppes of the Kirghiz among cattle and sheep, and which was supposed to be scarlet fever (?) by Petrowski.

The Plague (*Bubonic plague*) of man passes to pigs, rats and mice in China and Hong-Kong (Janson). The plague bacterium discovered by Kitasato and Yersin (*bacillus pestis bubonicæ*) is especially transmitted by affected rats and mice. These animals are the most active agents of the transmission of the disease to human beings. According to Yersin, cattle, horses and buffaloes, as well as rabbits and guinea-pigs, are susceptible to the disease. Carnivorous animals, on the other hand, appear to have but slight disposition thereto; birds are apparently immune. Robertson has observed a case in a cat, though Mattei and Simondi consider the cat to be fairly immune.

ADDENDA.

THE TERMS INFECTION AND CONTAGIUM.

BY M. H. HAYES, F.R.C.V.S.

SINCE ancient times, many attempts have been made to classify certain diseases which, respectively, have a more or less uniform type, and which are set up in the animal body by causes that cannot be perceived by the unaided senses of man. They have been divided, according to their respective modes of occurrence, into *epizoötic*, *enzoötic*, and *sporadic diseases*, which divisions are neither distinctive enough nor sufficiently well defined to merit adoption. Some of the diseases in question were found to be communicable from one animal to another by direct or mediate contact, and were consequently said to be "*contagious*" (Lat. *contagio* = contact); while others, such as bovine pleuro-pneumonia and rinderpest, having demonstrated their ability to go from one host to another independently of contact, were termed "*infectious*." This attempted classification was mainly one of degree: for the large majority of the so-called infectious diseases (such as foot and mouth disease and variola) are also contagious. As instances of the few purely contagious diseases, I may mention rabies and dourine. Besides, the distinction, as regards the mode of transference, between a so-called contagious disease and a so-called infectious disease, is often more apparent than real; for, as cases in point, the dried sputum of tuberculosis, or the dried bacilli of tetanus, may, on being carried by the air in the form of dust, set up their respective diseases in a manner which might appear to an unscientific observer as "infectious"; although the transference of the virus is as mechanical as that by the lancet or inoculation syringe. With advance of knowledge, so many of these so-called contagious and infectious diseases were proved to be caused by special micro-organisms, that pathologists felt justified in including among them a large number of similarly behaving diseases (such as distemper and variola), the exciting causes of which remain still unknown. As these microbic and supposed-to-be-microbic diseases had

the common property of possessing a contagium, or virus, by means of which all these diseases had been proved to be, or were supposed to be, propagated, whether from outside or from one animal to another, a classification founded on this property seemed to be as reasonable as it was convenient. The special meaning attached to the word "virulent" precluded the use of the adjective derived from "virus." The necessity of avoiding ambiguity prevented their being termed "infectious diseases," than which "contagious diseases" was a still more objectionable designation. The not very happy escape out of this difficulty has been to give them the name of "*infective diseases.*" An infective disease, taking for granted its microbic origin, may be defined as one that is caused by a living micro-organism which is capable of becoming developed in the animal body.

Although the condition of minuteness is purely arbitrary, it is well recognised in practice: for example, we withhold the term "infective" from mange, which is set up by an acarus; but we concede it to surra, which is caused by an infusorian. The tendency to become general is in most cases considered to be a characteristic of infective diseases; parasitic ringworm, which is due to a microscopic mould, being classed as a skin disease.

The words "*volatile*" and "*fixed*" are employed with reference to the capacity the contagium or virus has or has not of being carried to a distance from its place of development, whether such place is outside or inside the animal body, and independently of the mode of disease transference from one animal to another. Thus the virus of that non-contagious disease, South African Horse Sickness, is fixed; and that of malarial fever, which is a non-infectious disease, is volatile.

When the exciting cause of a disease can, like that of anthrax or tetanus, exist independently of the animal body, we apply the expression *ectogenous* to it. The cause of an *endogenous* disease, on the other hand, is one which, like that of glanders, can fulfil its life mission only in its animal host. Hence, in the case of an endogenous disease, the destruction of all infected animals, with the allowance of a reasonable length of time for the death of the special organisms, would prevent its further occurrence; but such action would have no such result with an ectogenous disease. A *miasmatic disease* is an ectogenous disease, the contagium of which is volatile.

NOTES ON BACTERIOLOGY.

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These Notes are intended to assist those readers of this book who are not conversant with bacteriological methods, to understand the bacteriological portion of the text. They are in no sense intended to be a detailed statement on the modern methods of bacteriology.*

Classification.—Pathogenic microbes belong either to the vegetable or to the animal kingdom. The pathogenic organisms belonging to the latter are certain protozoa, such as the malaria parasite of man, and analogous parasites (*Proteosoma*) of birds; the trypanosomes, flagellated forms, causing tsetse-fly disease, surra, and dourine of horses, and other diseases of cattle, also sleeping sickness of man; the *Piroplasmata*, causing Texas fever and red-water of cattle and sheep; and the *Coccidia*, cell parasites, such as that of the rabbit.

Pathogenic vegetable microbes, which form the vast majority of disease-producing germs, belong to the *Protophyta* (plants that have no sexual reproduction), and include: (1) many *bacteria*; (2) a few *yeasts* (*Blastomycetes*). A few *moulds* also are pathogenic. Although yeasts and moulds play a large part in the functions of animal life, few of them are actual disease

* There are many excellent manuals on bacteriology. Among the smaller works, *A Manual of Bacteriology*, by Muir and Ritchie; *A Manual of Bacteriology*, by Professor R. T. Hewlett; *Essentials of Practical Bacteriology*, by H. J. Curtis; *Bacteriological Technique*, by J. W. H. Eyre, are all excellent and reliable. The veterinarian concerned with the examination of milk may find *Bacteriology of Milk*, by Swithinbank and Newman, a useful book.

producers. As instances to the contrary, certain pathogenic yeasts, *Oïdium albicans* and the fungus of favus (a mould) may be cited. *Moulds* consist of branched filaments (*hyphæ*), which form a felt-work (*mycelium*) by becoming interlaced. *Sporangia* containing spores become developed at the ends of the threads (*hyphæ*).

Bacteria, yeasts and moulds are distinguished from one another by their respective *methods of multiplication*. Thus, bacteria multiply by division; yeasts by budding and by sporulation; and moulds by division, by branching, and by sporulation.

The Protophyta belong to the Thallophyta (plants that are not differentiated into stem, root, leaf, etc.), which are a sub-order of Cryptogams, the vegetable kingdom being divided into Cryptogams (flowerless plants) and Phanerogams (flowering plants).

For microscopic purposes, bacteria are usually classified as follows :—

1. Rods (*Bacilli*) whose length is greater than their breadth and whose sides are parallel.
2. Spherical cells (*Cocci*).
3. Spiral forms (*Spirilla*, *Vibrios*, and *Spirochaetæ*), which are curved rods that, on uniting, form wavy S-shaped, chain-like, or spiral threads (filaments).

These forms frequently occur in some characteristic grouping, and these different arrangements and groupings are respectively peculiar to different species. For example, a *diplobacillus* is formed by the transverse division of a bacillus into two parts which continue to remain together; *leptothrix* consists of long rods or threads which have no branches; and *cladothrix*, of long rods or threads which have branches. A *micrococcus* is a single small round cell; the *diplococcus*, two divisions of a single coccus with or without a capsule (as in Diplococci of Pneumonia and Gonorrhœa); a *streptococcus* is a chain of cocci each more or less a part of its neighbour, in longer or shorter chains, and sometimes in rosaries, as in Streptococcus of Pus and Erysipelas. The term *staphylococcus* (Gr. σταφυλή, a bunch of grapes) is applied to micrococci which occur in irregular masses, *e.g.*, the various Staphylococci of Suppuration. *Tetracocci* are packets of cocci in one plane, and *sarcinæ* are small cutical masses of cocci produced by division into four parts in two planes at right angles to each other. Again, we have different elements, or different arrangements of elements, having further charac-

teristics—*e.g.*, many of the bacilli have terminal or lateral *flagella*, which are long or short threads of protoplasm, not staining readily, and by their vibration causing progressive movement of the bacillus (*e.g.*, *Bacillus typhosus*). There is another marked difference which must always be borne in mind, namely, that due to polymorphism, which, amongst other things, gives rise, under abnormal conditions, to *involution forms*. These are degenerate forms and assume various shapes and sizes, diverging in a high degree from the normal. All these characters and modifications may and do serve as bases for classification. A convenient general classification is into the Lower Bacteria (cocci, bacilli, and spirilla), and Higher Bacteria (Leptothrix, Cladothrix and Streptothrix—one of the most important of which group is the *Streptothrix Actinomyces*).

The term *bacterium* (Gr. βακτήριον, a small rod) is now used, especially in the plural, as a generic name to cover all the micro-organisms, whether rods or round cells, and the term *bacillus* is confined to those which are strictly rods.

Bacteria may be divided according to a variety of characters or conditions. If they be classified according to their condition of growth in respect of oxygen, the classification would be as follows :—

1. *Aërobes*, which, like *Bacillus subtilis*, require air (oxygen) for the maintenance of their life.

2. *Anaërobes*, which, like the bacilli of tetanus, malignant œdema and Black Quarter, do not develop in the presence of free oxygen.

Facultative anaërobes grow moderately well under a free supply of air, or when oxygen is entirely excluded. To this group belong most pathogenic organisms. *Obligatory* aërobes cannot, or can only to a very slight extent, grow without oxygen. *Obligatory* anaërobes are unable to grow, or only feebly, unless oxygen is excluded.

If, on the other hand, bacteria be divided according to their method of nutrition, the classification would be as follows :—

1. *Parasites*, which feed on, or at the expense of, another living organism (animal or vegetable), and which may be divided into (*a*) *obligate parasites*, and (*b*) *facultative parasites*. The former, like the bacillus of leprosy, can grow only in their animal host. The latter, like the bacillus of anthrax, are able to vegetate both in the animal body and apart from it. Most pathogenic bacteria are more or less true parasites.

2. *Saprophytes*, which obtain their nutriment from dead organic matter, play an important part in the economy of nature in breaking down complex substances into simpler compounds, which may again be taken up and assimilated. Thus carbon and nitrogen are supplied to the higher vegetation which is necessary for life in the animal kingdom. Hence the majority of bacteria, being saprophytes, must be looked upon as essential to life. In a limited sense all the parasitic bacteria which can be subcultured outside the body are saprophytic.

Micro-organisms may also be divided into pathogenic (or disease producers) and non-pathogenic; or according to the predominant changes they produce, *e.g.*, nitrifying, lactic acid forming, chromogenic, etc.

Biology.—Bacteria are composed of a body of more or less homogeneous protoplasm, though many substances may be found therein. Sulphur, fat, starch, protein granules, and, in some organisms, pigment have been observed. Amongst the last-mentioned may be cited *Spirillum rubrum*, *B. prodigiosus*, *B. pyocyaneus*, and *B. violaceus*.

In addition, small granules may frequently be detected in the body of a bacillus, which, owing to their staining propensities, are called *meta-chromatic granules*. They should not be confounded with *polar bodies*, which are occasionally found at the extremities of bacilli or spirilla, and which, in their turn, must not be mistaken for *spores*. The bacterial cell is enclosed in a delicate sheath or cell-membrane composed as a rule of protein, and not of cellulose, as is generally the case with vegetable cells.

Bacteria are reproduced by fission or spore formation. The micrococci multiply by simple division, and, generally speaking, bacilli do the same, though they never divide longitudinally. The method of fission is simply that a slight indentation occurs which becomes a more and more marked constriction up to the point of separation. "Spore Formation" may be either inside the capsule of the bacillus—*endospore*—or outside as part of the chain of cells—*arthrospore*. Endospore formation is well seen in the Bacillus of Anthrax. The protoplasm becomes granular and the small specks enlarge into encapsuled, oval, sharply-defined bodies growing at the expense of the protoplasm of the bacillus. Eventually, either by exhausting the bacillus or bursting its capsule, the spore escapes. It fulfils its function like a seed, not producing other spores but germinating into a small bacillus,

The capsule or the modified protoplasm of the spore, or both, afford it a very high degree of protection from inimical influences, such as desiccation, heat, or chemicals ; especially is this the case with the spores of Anthrax, which can retain life and virulence in a dried condition for months and even years. Spores may occupy different positions in the bacillus in different species—for example, in the bacillus of Black Quarter, the spore is situated near one end of the rod. We have truly *terminal* spores in the bacillus of Tetanus, which then simulates a drumstick or pin. Again, though bacilli of which Anthrax is the type, have spores of a less diameter than themselves, the bacillus of Black Quarter produces a spore which is of greater diameter than itself and which bulges out the bacillary capsule. It is important to note that spore formation is not so much a multiplying stage as a resting one. One individual does not produce more than one spore and that spore produces only one bacterium.

Bacteria are motile or non-motile. The motility is due to delicate threads of protoplasm termed *flagella*, which may be either lateral, or terminal, or both, as in *Bacillus typhosus*. All movement is accelerated by heat, and inhibited by cold or colouring reagents.

In addition to true motility, all bacteria suspended in a fluid exhibit oscillatory movements of a physical character and known as "Brownian movement." All bacteria require moisture for development and suitable food material. Temperature also profoundly modifies bacterial development ; cold inhibits growth, until at, near and below the freezing point all growth ceases Heat, on the other hand, within limits, favours growth. For most species there is an *optimum* temperature at which they grow best ; some develop between 20° and 30° C., the pathogenic forms between 30° and 40° C., etc.

At temperatures above 40°–42° C. growth usually ceases, and most sporeless organisms are destroyed in a few minutes at 60°–65° C. Many substances, known as *germicides* or *antiseptics*, inhibit bacterial development or destroy vitality, *e.g.* carbolic acid, mercuric chloride, and many other metallic salts, etc.

Products of Bacteria.—The following are among the chief products of bacterial life :—

1. *Pigment.*—Many species produce vivid pigments, pink, red, yellow, violet, etc. These "chromogenic" organisms as a general rule possess little or no patho-

genic action. Light and oxygen are generally necessary for the production of the pigment.

2. *Ferments*, such as the peptic ferment, or that which splits up sugar, or that which coagulates casein in milk.
3. *Gases*, such as carbonic acid, hydrogen and sulphuretted hydrogen.
4. *Phosphorescence*, which occurs mostly in sea-water organisms.
5. *Chemical Poisons.*—These may be of the nature of alkaloids, the so-called ptomines, which occur in putrid food, tinned foods which have undergone putrefactive changes, etc. Generally they are bodies of complex nature allied to the proteins and are known as toxins. The toxin may be excreted by the organism, as in the case of tetanus, or it may be an integral part of the bacterial cells, as in the case of cholera, etc. The toxin may be formed not only in the body, but also in artificial culture, and it may be a direct secretion or the result of enzyme action.

In the causation of disease by micro-organisms there are therefore these two main factors: *the living organisms*, multiplying and passing in many cases throughout the body; and *the chemical poisons* produced by them, which act both locally and generally, and react again upon the organisms which have been their source. In some cases the organism remains more or less localised as in Tetanus and Diphtheria, and from the local site of infection produces its toxins, which by means of the circulation are carried throughout the body, bringing about the clinical symptoms and signs of the specific disease they represent. In other cases, like Anthrax, the bacilli themselves pass to all parts of the body producing their poisons. It is true the organisms and poisons, either separately or together, may and do cause tissue change. As bacteriology has advanced, emphasis has in a marked manner been placed less upon the organisms *per se*, and more upon their products. The action of bacteria, as mechanical irritants, plays a very small part in the processes of disease.

In any infection, both local and general effects may occur. The former are of the nature of inflammatory reaction, acute or chronic. Sometimes the lesion has a special site, as in Typhoid Fever or Diphtheria; in other cases, like Tubercle or Anthrax

it depends upon the point of inoculation. The final result in the tissue change will of course depend upon the dose of bacteria, as regards quantity, quality and specificity, and also upon the resistance or otherwise of the tissues ; it may be a comparatively transient inflammation or actual degeneration and necrosis. The toxins produced by pathogenic bacteria bring about the specific and general characteristics of the disease, *e.g.*, in Diphtheria or Tetanus. These toxins are not necessarily mere secretions of the bacteria ; but may result from the action of ferments produced by the bacteria.

It may be well here to remark that the association of germs with disease is not a recent discovery. Long ago they were found to be present in Anthrax and other diseases, but their mere presence in the blood or tissues is not of course sufficient evidence that they are the actual cause of disease. It is now generally agreed amongst bacteriologists that the following conditions (Koch's postulates) must be fulfilled before any micro-organism can be held to be the specific cause of any particular disease :—

1. It must always be found when the disease is present.
2. It must be isolated, and cultivated outside the animal body.
3. The culture must, on inoculation, produce the disease.
4. The same micro-organism must be separated from the newly infected animal.

To which may be added :—

5. The occurrence of specific reactions between the micro-organism and the blood serum of the animal invaded, or immunised with it, bacteriolytic, agglutinative, etc.
6. The presence in the animal dead of the disease and in the cultures of the micro-organism of chemical substances of similar physiological action.

Channels of Infection.—The possible modes of infection are as follows :—

1. *Hereditary transmission*, by which organismal diseases are conveyed to the fœtus from the maternal circulation does occur, but is the exception. More often a *hereditary susceptibility to infection* is transmitted, *e.g.*, as is frequent in tuberculosis.

2. *Inoculation.* Many diseases are artificially inoculable, but few are spread by this means. One of the best examples is

Anthrax. The commoner methods of experimental inoculation adopted in bacteriological investigation are the following: (a) *intravenous*; (b) *intraperitoneal*; (c) *intra-ocular*; and (d) *subcutaneous*, generally in the groin, and used in tuberculosis as a test of the presence of the bacilli in milk, etc. *Spraying* fine solutions of organismal culture into the trachea or nostrils and *ingestion* of food contaminated with bacteria or their toxins are methods which are adopted occasionally.

3. By the *respiratory tract*: this channel of infection is supposed to be frequently the means of spreading anthrax, tuberculosis, diphtheria, scarlet fever, and similar diseases. Its consideration opens up the large question of micro-organic life in the air. We can only mention one or two of the outstanding facts respecting this wider question. Tyndall* was one of the first to record the common existence of bacteria in the air and their relation to wet and dry surfaces. Dr. Russell of Glasgow† applied Tyndall's researches to practical sanitation. He pointed out that moist surfaces do not give off solid bodies "either by evaporation or under the influence of air currents"; that the locomotion of micro-organisms in the air is passive, not active; and that all organisms, even those which are microscopic, obey the law of gravitation. If these three axioms are true, it is obvious that in still air surrounded by damp surfaces we have, practically speaking, no organisms at all. And approximately this is so. Applying these facts to infectivity through the air we may understand that mucus, discharges, wet excreta, wet sputum, and all other similar vehicles of bacteria will not, so long as they are moist, part with any virus to the surrounding air. Manufacturers act upon this principle in their treatment of anthrax-loaded material. Before being disturbed, a bale of infected hair is immersed in water and allowed to soak, and the bale loosened in order that the water may gain access to every part. The workers are not allowed to touch the hair until the bacillary dust becomes wet, and they are required to sort out the hair while it is in that condition. Thus a dangerous process may be conducted in comparative safety. When infective dust becomes turned into mud, it will not spread disease. It must be remembered, however, that in coughing, and sneezing, and even in speaking, fine particles of saliva are expelled and conveyed to some distance, and thus pathogenic organisms may gain

* *The Floating Matter of the Air.*

† *Jour. of Royal Institute of Public Health*, Sept. 1896, p. 407.

access to the air, *e.g.*, in pneumonia and tuberculosis, without desiccation of the medium.

Even supposing the air to be heavily laden with germs, it does not necessarily follow that they will obtain entrance to the human tissues. Woodhead has pointed out that adenoid tissue, if its cells are active, whatever its situation, is a means of protection, for the reason that it contains an enormous number of active cells which are capable of taking up large numbers of micro-organisms and destroying them. There is, as is well known, a ring of such lymphoid tissue surrounding the entrance to the trachea, and another round the entrance of the œsophagus in man and other mixed-feeding animals. Yet, as Woodhead has further pointed out, it is possible—especially perhaps in pigs—that this lymphoid tissue may act as a medium for the conveyance of the disease from the outer surfaces to the tissues beneath. When the medium has dried and dust has formed, it may become a source of infection, but the dry state is fatal to many organisms.

4. By the *alimentary canal.*—Water, milk, and foods are three of the commonest vehicles for bacteria. Glanders, typhoid, cholera, epidemic diarrhœa, tuberculosis, actinomycosis, anthrax and many other diseases may be conveyed in the food of man or animals. Infection by the alimentary canal is the rule in glanders, anthrax, typhoid, cholera, and perhaps tuberculosis. In some cases the organisms may be destroyed by the acid gastric juice, and infection may thus occur when the stomach is empty but not when active digestion is in progress.

In all bacterial diseases it is desirable to detect if possible the locality and mode of entrance of the specific organism to the tissues.

Methods of Bacteriological Examination.—Broadly speaking there are three chief means by which knowledge is gained respecting bacteria, viz.: (*a*) Examination by the microscope; (*b*) Cultivation on artificial media, and (*c*) Inoculation of animals. By the first means, *morphological* facts are obtained; by the second, something is learned of the *biology* of the organism; and by the third, facts are elicited respecting its *pathogenesis*. A few notes will be added on each of these lines of investigation.

I.—EXAMINATION BY THE MICROSCOPE.

Micro-organisms may be examined under the microscope* in their natural unstained condition, or stained in such manner as to demonstrate their form, etc.

(a) *Unstained specimens* are readily made by placing a drop of the fluid to be examined on an ordinary slide and superimposing a cover-glass; or if it be a particle of solid substance (such as a culture growth), it may be taken and mixed with a drop of tap water or 0.8 per cent. salt solution. Fresh specimens are examined in this way for determining the form, presence or absence of motility, characteristic grouping, etc.

(b) *Stained specimens.* The microscopical examination of bacteria is greatly facilitated owing to the fact that their protoplasm readily takes up basic aniline dyes.

A very small quantity of a fluid or pure culture, or blood, sputum, discharge, etc., to be examined is placed upon a clean cover-glass or slide preparatory to microscopic examination. Such a preparation is known as a cover-glass, or a smear, preparation.

* A good microscope is essential. It should have objectives of $\frac{2}{3}$ inch, $\frac{1}{6}$, and $\frac{1}{12}$ (oil immersion), and be provided with a substage condenser. A lens of $\frac{1}{12}$th inch focal depth is the usual power required for the study of bacteria, although in some cases a lens of a focal length of $\frac{1}{18}$th inch, or even stronger, is desirable. Streptothrix actinomyces, which belongs to the Higher Bacteria, is better seen with a power of $\frac{1}{6}$th inch than with one of $\frac{1}{12}$th inch. The principle of the immersion lens is the filling up of the space between the lens and the cover-glass with a material whose refractive index is the same as that of the lens, so that there will be no loss of illumination by the rays of light passing through media of different powers of refraction, while proceeding from the object to the lens. The power of a microscope varies not only according to that of the lens, but also according to the power of the eye-piece. Thus the magnifying power of a 1-inch objective in a Swift's microscope varies, according to the strength of the eye-piece and to the fact that the draw-tube is closed or extended, from 25 to 140 diameters; a $\frac{1}{6}$th inch objective, from 175 to 690 diameters; and a $\frac{1}{12}$th inch objective, from 385 to 1,627 diameters. As a high-power lens gives a picture which has comparatively very little "depth" of focus, it is necessary to place the object under examination in as nearly the same plane as possible. Hence the material to be investigated should be reduced to an *extremely* thin film. The object should also be in the optical axis of the instrument, and secured in position by means of the spring clips. In using the oil immersion lens the body tube of the microscope must be screwed down until the lens is in contact with the oil, and nearly touching the cover-slip, final focussing being effected with the fine adjustment. The substage condenser must also be focussed so that the best illumination and visual picture are obtained. The best light must be obtained by adjustment of the mirror. A skilful use of the microscope depends, of course, upon an understanding of its parts, and upon practice.

By means of a platinum needle, sterilised in the flame, a minute particle of the material to be examined is spread over the cover-glass or slide in a very thin film, using if necessary a drop of water or salt solution. The cover-glass is then taken up with a pair of forceps and passed, with the film side upwards, through a Bunsen flame, in order, by coagulating the albumen, to make the material adhere firmly to the cover-glass. By this means the film is *fixed*. Two or three drops of the filtered staining solution (*e.g.*, gentian violet, methylene blue, fuchsin, or any aniline dye) is placed on the film, which in two or three minutes is rinsed in water, dried in the air, and mounted in Canada balsam. Such is the method of simple or single staining.

In the case of tissues, small particles may be teased up and smear preparations made, or preferably, thin slices or sections may be prepared with a microtome, after freezing or embedding in paraffin wax. Such sections may be stained in methylene blue and treated for mounting as detailed below for Gram's method.

It may be advantageous to use methods of staining which have the advantage of contrast colouring, staining the organisms one colour and the surrounding tissue or cells another. There are many methods for doing this; but the three simplest are those of Leishman, Gram and Ziehl-Neelsen, which may be shortly described as follows :—

The *Leishman method* is a particularly serviceable one for smear preparations of pus, blood, organs, etc., but not for sections. The preparations, *not fixed by heat*, are flooded with the Leishman stain (a double methylene-blue-eosin stain dissolved in methyl alcohol) for 2–3 minutes, distilled water is then added, and the staining continued for a further 5–8 minutes; the preparation is finally well washed in distilled water, dried and mounted. Bacteria and cell nuclei are stained blue; red-blood corpuscles, fibrous tissue and cytoplasm of cells, etc., pink. Protozoa such as trypanosomes are also well stained by this method.

In staining cover-glass, slide, or smear preparations by *Gram's method* the following procedure is adopted: The preparations are prepared, dried and fixed as described above (smear preparations of pus, blood, organs, etc., are better fixed by immersing in a mixture of equal parts absolute alcohol and ether for 10 minutes : *no* heat). The solutions required are (1) anilin gentian violet*; (2) iodine solution*; (3) alcohol,

and (4) for counter staining smear preparations, sections, etc., eosin.* The staining procedure is then as follows :—

1. Allow two or three drops of the gentian-violet stain to fall upon the cover-glass or slide and remain in contact with the film for *five minutes.*

2. Drain off the stain and treat *with the iodine solution* for 1–2 minutes.

3. Drain off the iodine solution, and treat with alcohol until no more stain comes away (usually 20–40 seconds).

4. Wash in water, blot off superfluous water, and set aside to dry. If thought desirable, the preparation may be counter-stained by the application of a very weak solution of fuchsin or with eosin.

In employing *Gram's method* for staining tissue sections, the section, if not fixed to the slide, is passed from watch-glass to watch-glass containing the solutions by means of a glass needle. If a paraffin one and fixed to the slide, the paraffin is removed by xylol, and the xylol with alcohol. The section is then flooded with the various solutions. In either case the section is stained with the anilin gentian violet for ten minutes ; after which it is treated with the iodine solution until it is the colour of a dry tea-leaf (blue-black), which generally takes about two minutes. After this it is treated with alcohol until decolourised. When the colour has left the section to a sufficient extent, it is flooded with the eosin solution for half to two minutes, after which it is treated with alcohol for 15–30 seconds and finally with xylol for 1–2 minutes. A drop of Canada balsam is dropped on the section, and finally a cover-glass is superimposed. In the case of a free section, it is lifted out of the xylol with a section lifter and carefully arranged on a slide and the Canada balsam dropped on. The parts played by the fluids in the Gram process are as follows : The anilin-gentian-violet stains the bacteria purple ; the iodine fixes the colour in the micro-organism, creating a chemical combination and forming a blue-black pigment ; the alcohol decolourises everything except the bacteria ; the eosin stains the tissue a light pink, and thus produces a counter-stain ; and the xylol clears the preparation.

•Only some organisms stain by the Gram method, not others

* Anilin gentian violet is made by adding one part of a saturated solution of gentian violet in alcohol to ten parts of anilin oil water. The iodine or *Gram's solution* has the following composition :—Iodine one part, potassium iodide two parts, distilled water 300 parts.

Eosin may be a 1 per cent. aqueous or alcoholic solution.

Tubercle, anthrax, actinomyces, tetanus and pyogenic cocci, yeasts and moulds are Gram-positive; glanders, typhoid, cholera, spirilla and protozoa are Gram-negative.

Ziehl-Neelsen's method is used for staining "acid-fast" bacteria. Most bacteria when stained with fuchsin are almost instantaneously decolourised when treated with 25 per cent. sulphuric acid. A few, however, retain their colour even after prolonged treatment with the acid, and these are termed "acid-fast." The principal acid-fast bacteria are tubercle, the bacillus of Johne's disease, leprosy, and others occurring in milk, etc. (see below). A modification of the method is also used for staining spores in bacilli. The carbol-fuchsin solution employed in this method is prepared by mixing

100 parts of a 5 per cent. solution of carbolic acid,
1 part of fuchsin,
10 parts of absolute alcohol.

The various stages of this process for staining a tissue *section* may be described as follows :—

1. In the case of an unfixed section, immerse in a watch glass of the filtered stain. For a paraffin section fixed to the slide, treat with xylol and alcohol (as in the Gram process), and then flood with the filtered stain. Then carefully warm the watch-glass or the slide on a metal plate or piece of asbestos cardboard until the stain steams. Allow the warm stain to act for 5–10 minutes.

2. Rinse off the stain with water and then treat the section with a 25 per cent. solution of sulphuric acid or of nitric acid, to decolourise it. In the acid the tissue loses its bright red colour and turns a pale yellow. Alternately remove it from the acid to the plain water and back again until it is of *a faint pink colour.*

3. Wash well to remove any of the superfluous acid.

4. Counterstain with an aqueous solution of methylene blue for one to two minutes.

5. Wash with water.

6. Treat with absolute alcohol for 20–30 seconds in order to dehydrate.

7. Clear with xylol.

8. Mount in Canada balsam.

For staining by the Ziehl-Neelsen method an ordinary film proceed as follows :—

The film or smear is prepared in the usual manner and fixed by heat or with alcohol and ether. Then stain as follows :—

1. Flood with carbol-fuchsin stain, and warm until it steams either over the flame or upon a warm stage. Stain for 3-5 minutes, replacing with fresh stain any loss by evaporation.

2. Wash well in water, and treat with 25 per cent. sulphuric acid, or 33 per cent. nitric acid, until the film remains decolourised when washed with water.

3. Dry between layers of fine filter paper, and counter-stain with Löffler's alkaline methylene blue.

4. Wash thoroughly, dry, mount, and examine.

If milk is under examination it should first be centrifugalised.* The centrifuge tubes must be thoroughly cleaned and films made from the deposit from at least 50 c.c. of milk centrifugalised at high speed for not less than half an hour. Stained by this method, the bacilli of tubercle or other "acid-fast" organisms will be stained red, and the milk or casein cells, and other bacteria, blue. It is exceptional for the tubercle bacillus to be detected by this method.

It is important to recognise that the non-pathogenic "acid-fast" bacilli will be stained by this process as well as the pathogenic tubercle bacillus. A number of different acid-fast organisms have now been isolated from milk, butter, grass, soil, dust, manure, etc. As this point is of great importance in differential diagnosis, it may be desirable to refer briefly to the subject of these "acid-fast" bacilli. The chief points for differential diagnosis between this group and the true tubercle bacillus are five:—(1) The tubercle bacillus shows a fairly uniform manner of growth; (2) it requires incubation temperature for growth in culture media; (3) it is unique with respect to its excessively slow growth; (4) it is as regards growth and propagation a parasite; and (5) on inoculation it produces pathological cellular changes distinct from the nodular new growths following inoculation of acid-fast bacilli. In a sentence, the acid-fast bacilli differ from the tubercle bacillus in three main particulars, viz.: morphology, conditions of development (chromogenicity, rapidity of growth, and wide range of temperature within which they flourish), and their feebler pathogenic properties. From these facts it follows that

* If no centrifuge is available the milk must be placed in a sterilised conical flask in the following manner: to 50 c.c. of milk set for sedimentation, 10 c.c. of liquefied carbolic acid crystals are added. The mixture is thoroughly shaken and poured into a tall conical glass. After standing for twenty-four hours a little of the sediment is taken by means of a sterilised pipette and the film made.

however great the degree of similarity between these various acid-fast bacilli, and however much it is possible by artificial cultivation to modify the morphology of the various forms, there is sufficient difference to enable a differential diagnosis to be made if all the biological characters are ascertained, and particularly the pathogenic properties. Hence the importance of applying the inoculation test to all acid-fast and tubercle-like organisms detected in milk or butter. *The pathological differences* from Koch's bacillus are that inoculation with acid-fast bacilli gives rise to no "giant-cells," no epithelioid cell clusters, and no tuberculous caseation. Nodular lesions occur suggestive of tubercule, but according to Potet, and Abbot and Gildersleive: (*a*) they constitute a localised lesion only, having no tendency to dissemination, metastasis, or progressive destruction of tissue by caseation; (*b*) they tend to terminate in suppuration like ordinary abscesses; (*c*) when occurring as result of intravenous inoculation they appear in the kidney, rarely in the lung and other organs; (*d*) the form of granuloma set up is similar to that of actinomycosis. This group of organisms is one of considerable importance to the milk bacteriologist, and in all investigations dealing with the tubercle bacillus, or with milk and its products, it is essential that the acid-fast bacilli met with should be clearly differentiated from the tubercle bacillus. Not sufficient care has been taken in this respect in the past. Any such organism found should be compared in cultural and pathogenic properties with the tubercle bacillus, and the various acid-fast organisms, and not simply accepted on tinctorial properties as being the tubercle bacillus.

Many organisms produce tubercle-like nodules, *e.g.*, the *B. pseudo-tuberculosis* of Pfeiffer, which occurs in guinea-pigs. It is, however, not acid-fast.

The Staining of Spores.—The following are the methods commonly adopted:—

Möller's method.

(*a*) Prepare the film as usual, fix and dry.

(*b*) Treat with alcohol for two minutes, and then with chloroform for two minutes; wash in water.

(*c*) Treat with chromic acid, 5 per cent. aqueous solution, for from one to two minutes; wash and dry.

(*d*) Stain in a watch glass of filtered carbol-fuchsin, warm gently till it steams; allow it to act for ten minutes and wash off with water.

(*e*) Decolourise cautiously with sulphuric acid (1–2 per cent.), and rinse in water. This should remove the carbol-fuchsin from the bacilli but not the spores.

(*f*) Dry and counter-stain with Löffler's blue for one minute. Wash off stain, dry, mount and examine. The spores will be stained red and the bacilli blue.

Simple method.

(*a*) Prepare the film as usual, but fix by passing twelve times through the flame.

(*b*) Stain the film as in Möller's method.

(*c*) Decolourise *cautiously* with 1 per cent. aqueous solution of sulphuric acid, or alcohol 2 parts, acetic acid 1 per cent., 1 part.

(*d*) Counter-stain with Löffler's blue.

(*e*) Wash, dry, mount and examine.

Capsule Staining.—*McConkey's Method.*—1.5 gram methyl-green crystals and .5 gram dahlia are rubbed up in 100 c.c. distilled water: add 10 c.c of saturated alcoholic solution of fuchsin, and make up to 200 c.c. with distilled water. Treat with stain for five minutes or longer, and wash *thoroughly* in a stream of water. The stain should be allowed to stand for a fortnight before use, and must be kept in a dark place.

By these or other methods of staining, specimens are prepared for the microscope. The microscopical examination yields information as to form, size, arrangement, spore formation, motility, etc.

2.—Cultivation on Artificial Media.

Various media, as soils for these microscopic plants to grow upon, are found to be necessary. Some of the commoner ones are broth, gelatine, agar, blood serum, milk, potato, and many special media for particular purposes. The property possessed by agar, which is prepared from Japanese seaweed, of remaining solid at a temperature of 40° C., and above, renders it specially useful for the cultivation of bacteria at temperatures above the melting point of gelatine (about 25° C.). There are many appliances which belong to a well-equipped laboratory which need not here be mentioned. It may, however, be desirable to state shortly what procedure is generally followed,

in order to place the reader in a position to appreciate more fully much that has been mentioned in the text of this book.

A cultivation must be made on some selected medium in order to isolate one organism from another, and so obtain what is called "a pure culture" and also to secure valuable data in the way of characteristics of growth, in short, to allow the organism to exhibit its own biological character. To obtain pure cultures it is necessary to adopt Koch's *plate culture* method, or some modification thereof. This process is shortly as follows:—

A small quantity of the fluid or substance to be examined is mixed with ten or fifteen cubic centimetres of liquefied gelatine, which is then poured out into a flat "Petri dish." The gelatine (sterile) solidifies at a low temperature, and the plate is incubated at room temperature. In the course of a day or two, signs of life appear in numerous points or "colonies," which represent individual bacteria, and are by this process of dilution separated from one other. It is now a simple process to examine these colonies by the naked eye and by the microscope to note their size, shape, configuration, consistence, colour, and arrangement in relation to one another, and whether or not they liquefy the gelatine. When these facts are ascertained, a sterilised platinum needle may be taken and colonies or parts of colonies removed and transplanted in agar, gelatine, milk, or upon potato or other medium, either as surface (or streak) cultures, or as stab (or puncture) cultures.

Surface cultures are made on the surface of a solid nutrient medium contained in a test tube, which has been sterilised and allowed to cool into a solid state while the tube was placed in a slanting position, in order to afford a large inoculation surface.

Stab cultures are made in solid nutrient media contained in test tubes. The inoculation is effected with a sterilised platinum needle on the end of which a minute portion of the colony is taken up, and the needle is inserted for about an inch or more in depth into the nutrient medium along the middle axis of the tube, under the usual precautions against contamination. If the organism under examination is an aërobe, it will grow only on the surface of the medium; if an anaërobe, only in the track of the needle; and if a facultative anaërobe, it will grow both on the surface and in the track. We may obtain valuable diagnostic information from the presence or absence of liquefaction, gas-formation, rapidity of growth, or pigment production;

and especially from the appearance of the growth in the track and on the surface. Moreover, when the organism is thus isolated in pure culture its further study is practicable.

The Diphtheria bacillus (*B. diphtheriæ*) grows very rapidly (12 to 24 hours), and appears as small scattered white colonies having a darker centre. Non-liquefying. The Anthrax bacillus (*B. anthracis*) produces in a stab gelatine an arborescent growth of fine branching threads running out horizontally from the track of the needle. Liquefying gelatine. The Cholera vibrio has a characteristic mode of liquefaction. Koch's comma bacillus produces one large bubble at the point of inoculation which slowly increases in size. Finkler-Prior's bacillus liquefies rapidly and results in a tube of fluid with the organisms collected in a mass at the bottom. The streptothrix of *actinomyces* produces a dry, rough, raised, crinkled brownish growth on agar, and a rugged, raised yellowish growth on potato. The Glanders bacillus (*B. mallei*) on glycerine agar forms a uniform streak of grey-white colour of a shiny consistence. On potato it shows a characteristic growth of a brown-yellow tint on the third day and a chocolate brown later. The Tubercle bacillus (*B. tuberculosis*) produces on glycerine agar in 2–6 weeks (it is one of the slowest growing of pathogenic germs) a raised, smooth, confluent growth which later becomes dry and irregularly crinkled. *Streptococcus bovis* appears in numberless, small, delicate white colonies, non-liquefying, and localised to the needle track. *Staphylococcus pyogenes aureus*, found commonly in pus, is a comparatively rapid liquefier of gelatine, and forms an orange growth on agar. The bacilli of Tetanus, Malignant Œdema, and Black Quarter, are types of anaërobic organisms, and the medium in which they grow is more or less split up by gas formation. The organism of Typhoid Fever (*B. typhosus*) and *B. coli* do not show such marked features in cultivation as some other organisms, but they produce characteristic changes in sugar media and possess biological features by which they may be differentiated.

The bacilli of Leprosy and of Johne's disease have not yet been cultivated artificially.

Anaërobes may be cultivated in an atmosphere of hydrogen or *in vacuo;* or various agents may be added to the media for removing the oxygen. There are a variety of methods for obtaining these conditions.

3.—The Inoculation of Animals.

It is evident that to test the powers of disease-producing which any organism may possess, it is necessary to inoculate animals (*e.g.*, guinea-pig, rabbit, mouse, etc.), to ascertain exactly what pathological effect is produced by the organism. A special licence is required in Great Britain for such experiments. It will be sufficient to remark that the simplest forms of inoculation are all that are usually required in milk investigation, namely, the *intraperitoneal* and the *subcutaneous*. In some cases it may be sufficient to inoculate a few c.c. of the original milk; but, as a rule, it is advisable to centrifugalise, or use the sedimentation flask containing about 250 c.c. From the deposit or sediment two guinea-pigs may be inoculated, the one subcutaneously in the groin, the other intraperitoneally. Particularly is this necessary in making a reliable and exhaustive search for the tubercle bacillus. *Microscopical examination alone for this organism is not reliable.* The details of the process as carried out in practice are as follows:—After centrifugalisation the deposit is mixed with the 2–3 c.c. of milk remaining in the tube after aspiration of that which is superfluous. Two guinea-pigs (of say 300–400 grammes weight each) are taken and inoculated with the deposit from about 100 c.c. of milk. The fluid is inoculated subcutaneously on the inner side of the thigh or lower part of the abdomen under aseptic precautions into one guinea-pig and intraperitoneally into the other. In three weeks' time, if the inoculated milk contained a considerable number of tubercle bacilli, typical infection of the popliteal and inguinal glands can be detected. If the milk contains very few bacilli the infection is much slower (5–6 weeks). After the animal has been killed, the presence of the tubercle bacilli can be detected in smears made from the infected glands and spleen. Some workers make it a rule to inoculate two guinea-pigs from the sediment of the milk, one receiving half of the sediment subcutaneously in the groin, the other receiving the remaining half intraperitoneally.

Routine Procedure in the Bacteriological Examination of Cows' Milk.—Physical examination (temperature, reaction, colour, cream, deposit, specific gravity, etc.) of the milk should be made if necessary. The microscopical examination of the sediment after centrifugalisation or sedimentation will detect the presence of dirt (*e.g.*, hair,

straw) and of blood-cells, excess of leucocytes and pus-cells, etc., and stained films of the sediment will show streptococci and other micro-organisms.

1. *Plate cultivation.*—Dilute with sterile water or broth as required (probably 1 : 100,000) and make plate cultivations in Petri dishes. Six or more gelatine plates should be made with quantities varying from 0.05 to 0.5 c.c. of the dilution and incubated at room temperature. Plates should also be made with nutrient agar for incubation at 37° C. Other media may also be selected. The plates should be counted on the second, third, and fourth days, and the necessary subcultures made. Agar plates incubated wholly at 18° or 22° C., will in the long run show more colonies than when incubated first at 37° C. and then at 22° C., or at 37° C. throughout.

2. *Anaërobic cultivation.*—At the same time that the primary aërobic plate cultivations are made, similar plates may be made on lactose-gelatine and lactose-agar for anaërobic culture.

3. *B. coli test.*—To seven tubes of McConkey's bile-salt lactose peptone water are added respectively, 10 c.c., 1 c.c., 0.1 c.c., 0.01 c.c., 0.001 c.c., 0.0001 c.c. and 0.00001 c.c of the milk. These should be incubated at 42° C. for two days, and the presence of gas and acid in any tube is presumptive evidence of *B. coli*, which must then be isolated and identified. By adding to several tubes of medium different quantities in this manner a *quantitative* result is obtained, *e.g.*, we may say *B. coli* is present in 1 c.c. but not in 0.1 c.c.

4. *B. enteritidis sporogenes test.*—100 c.c., 10 c.c., 1 c.c., 0.1 c.c., 0.01 c.c. and 0.001 c.c. of milk are taken. The 100 c.c. is placed in a large tube, the other quantities are added to tubes of sterile milk. All are heated to 80° C. for 10–15 minutes, and incubated anaërobically at 37° C. for 48 hours. Formation of a surface honeycombed clot of casein floating on a watery whey is indicative of the presence of this organism.

5. *Examination for special micro-organisms.*—The milk must be centrifugalised or the particulate matter allowed to gravitate by sedimentation. It is, as a rule, useless to attempt examination microscopically or otherwise without first using the centrifuge or sedimentation flask. The deposit is then to be stained for the particular organism for which search is being made.

For centrifugalisation, 200 c.c. of the milk under examination should be placed in the sterilised tubes of the centrifuge. In these tubes the milk may be centrifugalised for half an hour at 2,000 revolutions a minute. At the end of such a

period the milk in each tube has separated into three layers —at the top there is a dense layer of cream, at the bottom there is the sediment or "slime" containing all the particulate matter, and between these two is the separated milk. Aspirate off the cream by means of a sterile glass tube connected with an aspirator or vacuum pump, and examine separately; aspirate all the separated milk except 2 c.c. The remaining sediment is so compact and dense that the tube may now be inclined and the sediment fully exposed without displacement. By means of a sterilised platinum loop a small portion may be taken up and spread on the surface of half a dozen slides and stained. The remainder of the sediment is well mixed with the 2–3 c.c. of milk and used for inoculation of guinea-pigs.

For sedimentation take two conical sedimentation glasses and fill them with the milk under examination, allowing them to stand in the refrigerator for twelve to fourteen hours. It is customary to add a few small carbolic crystals to each flask (this, of course, must *not* be done if the sediment is to be inoculated into animals). On the completion of sedimentation the milk has separated into three main strata, the cream at the top, the sediment at the apex of the flask, and the separated milk in the middle. The cream and milk may be decanted, and the sediment examined for any special micro-organisms.

6. *Tubercle Bacillus.*—The inoculation of guinea-pigs is the only certain test for this organism (see above).

Immunity.—No fact in biology is more striking than the varying susceptibility to infective diseases displayed by various animals. Thus, while cattle and ordinary sheep are highly susceptible to anthrax, Algerian sheep, white rats, and birds are insusceptible. Such an insusceptibility to disease is known as "*immunity.*" Immunity may be "natural," *i.e.*, the ordinary condition, as, for example, the normal insusceptibility of the white rat to anthrax; or it may be "acquired," *i.e.*, the ordinary susceptible condition is converted by some event or treatment into an insusceptibility. Acquired immunity generally results either from an attack of disease ending in recovery, *e.g.*, animals which have recovered from rinderpest or Texas fever are no longer susceptible to the disease; or by vaccination, *e.g.*, in the case of animals vaccinated against anthrax or black quarter. In man, natural immunity is seen against many animal diseases, *e.g.*, black quarter and tsetse-fly disease of horses, and acquired immunity normally follows

an attack of small-pox or scarlatina, and may be induced by vaccination in the case of small-pox, cholera, plague, etc.

Immunity has also been classed as *active* or *passive*. Active immunity results from an attack of disease or after vaccination with an attenuated virus, as in the case of anthrax. Passive immunity results from the injection of the blood serum of an immunised animal. The former is of relatively long duration, and is due to an alteration in the fluids, cells, and tissues; the latter is of short duration, and is due to the "protective" substances injected, there is no alteration in the fluids and cells of the treated animal, and immunity lasts only so long as the protective substances remain.

The question arises, What is it that makes the blood and body tissues insusceptible to the disease? The following are the chief theories which have been advanced to account for this condition.

1. *The Theory of Exhaustion.*—This supposed that there were in the body certain substances or conditions which were necessary or at least favourable to the existence of some particular micro-organism and its products, and that when that particular organism had once been present and had extended itself throughout the system, it used up these favourable substances or conditions to such an extent, that when the patient was again exposed to the infection, his body no longer offered a favourable medium. This theory was also supposed to account for the fact that the disease died out in the first instance; the pabulum being exhausted, the organisms could no longer exist and the patient recovered. But there is no evidence that any such "exhaustion" occurs.

2. *The Theory of Retention*, the opposite of the foregoing supposition, assumed that the organisms produce substances which are inimical to their further growth. In a certain sense this theory has some truth in it.

3. *The Theory of Acclimatization* supposed an acquired tolerance on the part of the tissues to the toxic products of micro-organisms (*Sternberg*).

4. *The Theory of Phagocytosis.*—Metchnikoff advanced the theory that micro-organisms introduced into the body are got rid of by the action of amœboid or wandering cells, which ingest the microbes and destroy them. These cells—certain leucocytes, endothelial and other wandering cells—he termed *Phagocytes*. The chemical products secreted by micro-organisms as a rule exert an attractive influence on the phagocytes, so

be due to absence of the particular receptors necessary to fix the poison. Acquired immunity must be regarded as an alteration in the fluids and cells of the body and the various factors of bactericidal action, phagocytosis, and opsonins probably all play a part.

We may now consider the practical formation of antitoxins artificially. As we have already seen, bacteria produce disease by two main factors: first, by the multiplication of the living organisms in the body to which they have gained entrance; and, secondly, by the production of toxins. In the course of time these toxins are either transformed by the body cells, or, by their interaction with the body cells, by a process at present unknown, may produce antagonistic substances which are inimical to their own action.

To obtain these substances artificially for the treatment of cases of disease, a virulent toxin of the specific organism of the disease is inoculated into an animal, whose blood serum, in the course of time, will contain antitoxins. It is then drawn off, and used for inoculation as a protective or defensive agent. The details of the process may be best illustrated by taking a concrete example, *e.g.*, Tetanus :—

1. *The Preparation of the Toxin.* The tetanus bacillus is grown in glucose broth in hydrogen for 3–4 weeks, and the culture is then filtered through a Chamberland filter. Of the filtrate, $\frac{1}{1000}$ c.cm. ought to kill a guinea-pig (say of 250–300 grammes), and it is by estimating the lethal power in a standard guinea-pig that the toxicity is determined.

2. *Immunisation of Horse with Toxin.**—The toxin may at first be weakened before inoculation into the horse by the addition of Gram's iodine solution in the proportion of 1 : 3. Injection is made subcutaneously and intravenously in gradually increasing doses. An injection is given weekly for many weeks, until the horse is able to stand without injury a very considerable dose of toxin.

3. *Preparation of Antitoxin.*—The blood serum is from time o time tested by withdrawing a small quantity and observing ts power to neutralise a given amount of toxin (say ten lethal loses), when injected into a mouse or guinea-pig. When the equired degree of antitoxic power is reached, blood is drawn ff under aseptic precaution from the jugular vein, allowed o coagulate, and the serum separated. This is collected, and

* The horse is used on account of its size and its tractability, and because its rum is less toxic than that of other animals.

contains the antitoxin, which is now ready for use, and may be kept (preferably with a small percentage of carbolic acid or other preservative agent added) in solution or, by evaporation over sulphuric acid *in vacuo*, in the dried form. Serum yields roughly 10 per cent. of dried substances, and should be kept in a cool, dark place.

4. *Clinical Therapeutics.*—It is clear that the sooner the antitoxic serum is injected into the patient or animal suffering from Tetanus, the better The results of Diphtheria compare favourably with Tetanus in this respect, for the simple reason that in the former it is possible to diagnose the condition comparatively early, whereas in Tetanus the condition cannot be detected till in an advanced stage. The disease in the patient has, so to speak, *to be caught up*: it has had the start. The immunity conferred by the injection of antitoxin is but temporary, lasting only about three weeks; but clearly that is sufficient to tide over the acute stage. The same principles are employed for the production of toxin and antitoxin for therapeutic use in Diphtheria.

Antitoxin can also be used as a prophylactic, *e.g.*, in the case of operations on valuable animals.

In the case of organisms which produce no extra-cellular toxin, *e.g.*, anthrax, a true *antitoxic* serum cannot be prepared, or at best but a feeble one. Animals are immunised by the injection of sub-lethal doses first of killed, and then of living cultures, and after prolonged treatment the blood serum acquires some curative and prophylactic properties, but feeble compared with those of an antitoxic serum.

END OF VOLUME II.

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